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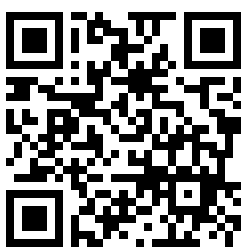

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THE

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THREE CASES
OF INTEREST IN CONNECTION WITH THE CÆCUM.

By J. BLAND-SUTTON, F.R.C.S.

CANCER OF THE CÆCUM.

IT is an interesting fact, clearly brought out in the recent cancer agitation, that in more than half the cases in which death is attributed to cancer the primary seat of the disease is in the digestive organs. The distribution of cancer in that part of the alimentary canal which occupies the belly is somewhat remarkable. For example, the stomach is not only the commonest primary seat of cancer when compared with other digestive organs, but it stands third in order of frequency among all organs. The breast (mamma) being first and the uterus second in order of liability. The small intestine (duodenum, jejunum, and ileum) is very rarely attacked by cancer, but in the rest of the canal (colon and rectum) it is a very frequent disease. Even in its attacks on the big bowel cancer exhibits a partiality at present inexplicable, for it is common to find it in the rectum and sigmoid flexure, but its appearance in the vermiform appendix and ileo-cæcal valve is phenomenal; and its presence in the cæcum may be fairly described as unusual.

It is as difficult to explain the comparative immunity of the ileo-cæcal valve from cancer as to find an adequate cause for its excessive frequency at the pylorus.

A careful study of the records of cancer occurring in the cæcum, as well as my own observations, leads me to the opinion that when care is taken to distinguish between cancer of the cæcum, the ileo-cæcal valve, the vermiform appendix, and the first segment of the ascending colon, it will be found that primary cancer of the cæcum is by no means common. It is on this account that I venture to record the subjoined case:

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A single woman æt. 55 years came under my care for a painful swelling in the right iliac fossa; it was movable, and about the size of a bantam's egg. Though this tumour felt firm or even hard, it yielded a resonant note on percussion, and I had no doubt that it was a tumour either of the cæcum or of the cæcal end of the ascending colon.

On June 2nd, 1902, I exposed the cæcum by a free incision in the right iliac region, and found it occupied by an indefinite hard tumour; and as the adjacent mesentery contained some enlarged and hard glands I detached the cæcum and vermiform appendix from their connections with the peritoneum, ileum, and ascending colon, and dissected the enlarged lymph-glands from the mesentery. A number of branches of the ileo-colic artery were secured with silk ligatures, and the cut edges of the mesentery were united with sutures of the same material. As the lower end of the colon was too big to accommodate the ileum, I narrowed its lumen by suturing its cut edges to the extent of one half its diameter. With the assistance of a small decalcified bone bobbin the cut end of the ileum was easily anastomosed into the ascending colon; peritoneum surfaces of ileum and colon were closely adjusted by interrupted sutures of silk. A thin gauze drain was introduced, and the wound secured with sutures of silkworm gut. Small quantities of gas escaped along the drainage track for about twenty-one days; then the wound cicatrised, and five weeks from the date of the operation the patient returned to her home.

The parts removed were hardened and then sectioned as shown in Fig. 1. Microscopically the mass exhibited the characteristic structure of cancer as usually found in the colon, but there was an excess of the connective tissue, or matrix element. A study of the specimen shows that the disease arose in the cæcum. It is quite in accord with what we know of primary cancer in other regions of the colon that the disease rarely gives rise to physical signs or distress until it is well advanced. This "latency" of cancer is a feature more marked in the case of the cæcum than in any other part of the digestive tract.

Acute Distension and Partial Rotation of the Cæcum.

In May, 1902, Dr. Albert Paling asked me to see a lady about thirty years of age, mother of two children, who, a few hours previously, had been seized with sudden and acute pain in the belly, followed by such alarming collapse as to suggest perforation of a gastric ulcer. This diagnosis was further supported by the fact that the patient had

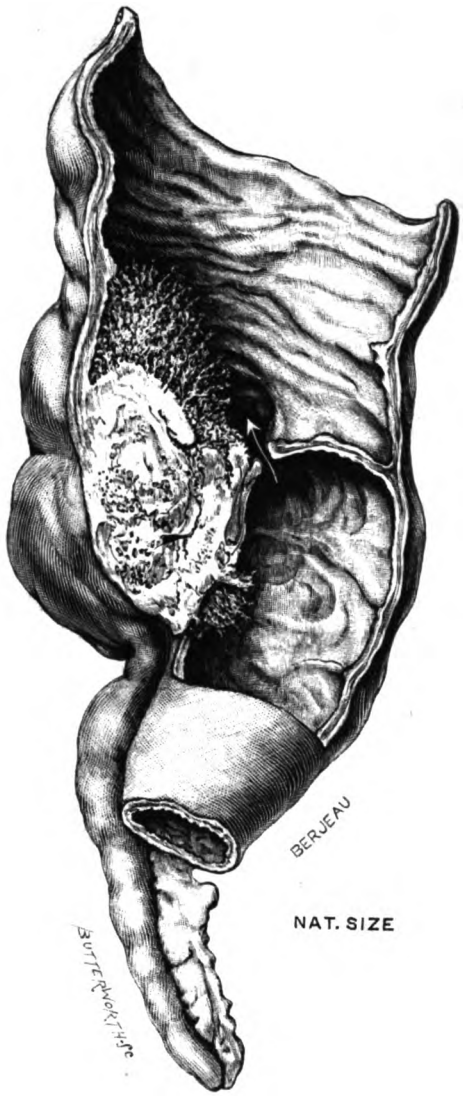

Fig. 1.—A cæcum with the vermiform appendix and adjacent segments of the ascending colon and ileum. The cæcum is occupied with a cancerous mass, which has perforated its mesial wall and not only implicated the adjacent segment of the ileum, but has penetrated its lumen. The parts were successfully resected from a woman æt. 55.

suffered from chronic dyspepsia. As the signs of collapse passed off the abdomen slowly distended, the temperature rose to 101°, and the pulse quickened to 120 per minute.

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From a careful study of the history of the case and a thorough physical examination of the patient I had no doubt that there was something very seriously wrong in the belly, but I could not satisfy myself as to the viscus at fault. Though the belly was everywhere distended and tympanitic, the distension could be described as lopsided, being much more marked in the region of the cæcum.

The patient's general condition being very serious, we had no doubt as to the propriety of cœliotomy. On opening the abdomen a large, globular, glistening, tense, translucent body presented itself; with a little care we determined this to be the cæcum, but the distension was so great that the peritoneum on the crown of this big gaseous cyst was split. I punctured the cæcum with a needle and allowed the gas to escape, and as the bowel collapsed we found the ileum entering it on the outer side, so that the cæcum had rotated on itself to the extent of nearly half a circle, and had, of course, carried the terminal section of the ileum with it. The distension was abruptly limited to the cæcum. After satisfying ourselves as to the relations of the parts I made a small opening in the collapsed cæcum, and secured it to the lower angle of the abdominal incision, for I felt that if the cæcum were left in its collapsed and paralysed condition, it might again slowly distend with gas if no channel were available for its rapid escape. The rest of the wound was closed with silkworm-gut sutures and silk in the usual way.

The convalescence was in every way satisfactory; the wound healed soundly, and no fæcal matter escaped through the temporary sinus.

I venture to record the case as it is a striking parallel to that severe and often fatal condition of the stomach known as "acute dilatation of the stomach," on which my colleague Dr. Campbell Thompson has recently published an important monograph.

A Dermoid of the Cæcum.

I have often stated that in women "dermoids have never been found growing primarily from any abdominal viscus save the ovary." The sheets of the second edition of my book on 'Tumours' were scarcely dry from the press when Mr. C. Hall, of Sheffield, informed me that he had found a dermoid connected with the cæcum, and he was kind enough to send the specimen for examination, and allowed me to have a drawing made of it (Fig. 2).

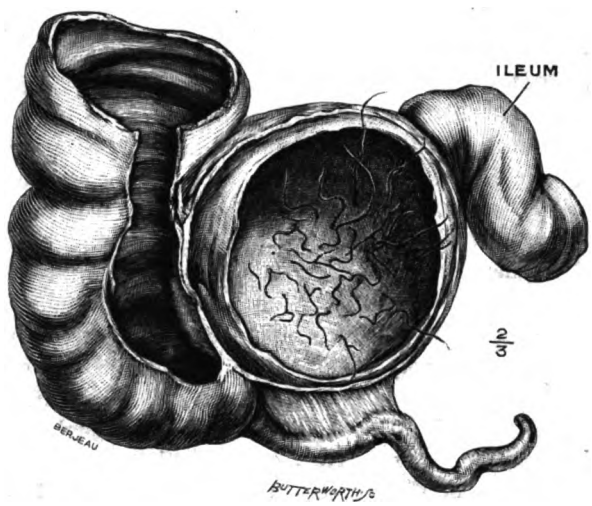

Fig. 2.—The cæcum and adjacent segment of the ileum. A dermoid occupies the angle between ileum and cæcum. (From a specimen in the possession of Mr. C. Hall, of Sheffield.)

This cæcum with the terminal portion of the ileum is from a man, and as far as we know it did not puzzle physician or surgeon, or in any way disturb the man, for the dermoid was accidentally discovered at the *post-mortem* examination. The position of the tumour is remarkable, for it lies in the angle formed by the junction of the ileum and

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cæcum, and, as is well shown in the drawing, it lies between the layers of the peritoneal fold, extending from the termination of the ileum to the mesentery of the vermiform appendix. It contained the usual pultaceous matter and hairs. On microscopic examination a lining of stratified epithelium was detected, but no stratum granulosum.

Morphology fails to assist us in speculating on its mode of origin. I have critically examined several hundred dermoids from human beings, and from all the recognised situations; I have hunted records in many languages for facts regarding dermoids; but I have never seen nor read a description of anything similar, and therefore venture to publish the case with the hope that others who may have seen the like will record their observations.

A Peculiar Case of Pneumothorax in a Man of 22.—The striking facts about it were that at the time of its onset the attack caused absolutely no symptoms. The man complained of having had slight pains in the right chest, and a little dyspnœa. All this had begun very gradually. There was no history of trauma, fever, or chill. He had had the same feelings about a year before. He had worked until the day of his admission into the hospital, and his symptoms were very slight indeed. There was no history of tuberculosis in the family; the patient had no previous history of any importance, and had no specific history. He was found to have a high grade of right-sided pneumothorax. A Röntgen-ray examination gave confirmatory evidence of the diagnosis. The man had improved very greatly after about six weeks' treatment, when he suddenly had a recurrence of exactly the same symptoms as at first, and again showed the physical signs of a marked pneumothorax—again on the right. The patient was discharged about three months after his admission, with his pulmonary physical signs practically normal. At no time was there any evidence of tuberculosis, and there was no history or evidence of previous pleurisy. The almost entire absence of symptoms was the most striking part of the case. The importance of the Röntgen rays in this case is insisted upon, particularly in the demonstration of the fact that pleural adhesions must have been practically entirely absent.—*Phil. Med. Journ.*, September 20th.

OBSTRUCTED BREATHING IN EARLY LIFE.

By G. A. SUTHERLAND, M.D.,

Physician to Paddington Green Children's Hospital, and to the City Orthopædic Hospital.

THE preservation of the respiratory passages in a healthy condition during the early years of life is a matter of the greatest importance for the well-being, it may be the life, of a child. Local disease in those passages is common, and may produce a weak spot at which an acute infection gains entrance to the body, or may, by mechanical obstruction to the respiration, lead to disease in vital organs and imperfect development of the system generally. In fact, obstruction to the air current may be said to be the important factor in respiratory diseases, just as obstruction to the blood-stream is in cardio-vascular disease. The cause of the obstruction may be (*a*) inside the air-passage, for example, a foreign body in the larynx; or (*b*) in its wall, as in bronchitis; or (*c*) outside its wall, as in the case of a tumour pressing on the trachea—the result in all cases being the same, namely, a diminution in the size of the air-conducting space at the seat of obstruction. Perhaps a fourth cause of obstruction ought to be mentioned as not exactly embraced by the other three, and as being a very common condition in early life, namely, (*d*) spasm of the muscles in the respiratory passages.

Taking the term obstruction of the respiratory passages in its widest sense, we observe that those passages begin at the nostrils and terminate in the alveoli of the lungs, and that obstruction may be in or around those passages at any part from the openings of the nostrils to the costo-abdominal parietes of the thorax. In order to guard as far as possible against obstruction, the air-passages are rendered non-collapsible by a strong framework of bone, muscle, and cartilage. In the nose and pharynx the supports are of bone and cartilage: in the larynx, trachea, and bronchi they are of cartilage; while in the case of the soft tissues of the lungs there is a perfect safeguard in the bony and cartilaginous walls of the thorax. We observe also how the respiratory tract is double at the beginning (the nostrils), and the end (the pul

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monary air-tubes), and is single in the middle (from the larynx to the bifurcation of the trachea), and that therefore the gravity of an obstruction in the single passage is much greater than in the case of the double passage. In the latter case it frequently happens that only one side is affected, and an alternate route, more or less sufficient for breathing purposes, remains patent. The symptoms also are, as a rule, much more severe in obstruction of the single passage than of the double. The contrast between the severe effects produced on the system by a sudden interference with normal action, and the toleration which can be established by a gradual interference is well illustrated in the case of obstruction in the respiratory passages. Taking it in its most typical form, namely, laryngeal obstruction, one observes in an acute case the anxious expression of the patient's face, the hurried breathing, the erect position, the restlessness, the silence unbroken by word, cry, or cough which can be avoided, the sweating, the dusky face with dilated cervical veins, the loud stridor, the expanding alæ nasi, the movements of the larynx, the heaving trunk with all the muscles standing out in powerful action, and the inspiratory retraction of the soft parts above the clavicles and the sternum, of the sides of the chest, and of the upper part of the abdomen. In the chronic form, on the other hand, while the stridor, the retraction, and the hurried breathing may be present the child is not distressed, but lies smiling or crowing or talking, and the signs of distress are only elicited temporarily under excitement or from a fit of crying.

Acute obstruction, as from the presence of a foreign body, laryngeal diphtheria, or retro-pharyngeal abscess, is, in the majority of cases, of a temporary nature, and although it may cause serious local disease, does not as rule produce any local or general developmental injury. In chronic obstruction, on the other hand, one sees the most serious consequences, both constitutional and local, from the imperfect entrance of air into the lungs. Children who have suffered from prolonged obstruction are stunted, flabby, and anæmic; mentally they are backward, sluggish, or almost imbecile; in disposition they are dull and apathetic; they are easily tired, bear illness badly, and are prone to catarrhal and other inflammatory affections of the respiratory tract. In marked cases one can frequently tell from the altered facial appearance or the shape of the chest what form of obstruction a patient has suffered from, even although the obstruction has passed off, or has been removed some time previously. Consequently the preventive and curative treatment of the various forms of respiratory obstruction met with in early life forms a most important branch of therapeutics.

a. The Nostrils.—The most important affections in this region are simple catarrh, syphilitic catarrh, foreign bodies, fibrinous rhinitis, hypertrophied mucous membrane, and a deviated septum.

1. *Simple Catarrh.*—It is very common to find that during the first weeks of life an infant's nostrils have become blocked as the result of a simple catarrh. Nature has been extremely economical here in the matter of space for breathing purposes, has lined the passages with a delicate and sensitive membrane, and has provided the possessor with but limited powers of keeping those passages clear. Consequently a very slight obstruction, for instance, some inspissated mucus, starts a catarrh, which, if not attended to at once, quickly becomes chronic, and may seriously interfere with the infant's feeding, sleeping, and normal development. It has, unfortunately, come to be regarded by many mothers as the normal condition of affairs that an infant's nostrils should be blocked or discharging, and "the snuffles" in the majority of cases is not the result of syphilis, but of the neglect which follows on this foolish belief. It will therefore secure much immediate comfort to the child, and prevent to a great extent the risk of future trouble, if it is made part of the daily toilette to clear out the nostrils with some non-irritating lotion. This may be efficiently done by inserting a fine camel's-hair brush dipped in a watery solution of common salt ($2\frac{1}{2}$ grains to the ounce) or bicarbonate of soda (5 grains to the ounce), the solution having first been warmed. A thin brush can be passed easily along the floor of the nose to the post-nasal space, and if sneezing is provoked it will only ensure more thorough clearance of the nostrils. After the washing out a little dilute ointment (zinc or boracic) may be introduced into the anterior part of the nostrils in a similar manner, and the nasal toilette is then complete for the day. The question next arises as to how long this process should be continued. I believe that it will with advantage be kept up during the first two years of life, and

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afterwards until the child has learned to blow its nose, and thus clear the passages.

By this preventive treatment many "colds in the head" in young infants will be avoided, and normal nasal breathing will be maintained. If an attack of acute catarrh takes place, the treatment will be somewhat different, and the sequelæ of such a condition are very important. An attack of acute nasal catarrh passes very easily into a chronic one, and that, again, in the lapse of time, is followed by many remote effects in different parts of the system from the obstruction to the breathing. Chronic catarrh means a chronically swollen mucous membrane of the nose, and this inflammatory condition spreads very easily into the naso-pharynx, where the soft tissues also swell up readily, and soon adenoid hypertrophy takes place. The nasal space being rendered insufficient for breathing purposes, mouth breathing, especially during sleep, is necessarily resorted to by the patient, and results in attacks of pharyngitis and tonsillitis, with chronic thickening. Coughing is now added to the other symptoms, and this will probably attract the attention of the mother sufficiently to induce her to think a bottle of cough medicine advisable. But the trouble still goes on, and the inflammatory process next invades the larynx, as indicated by the husky voice and "croupy" cough, and the trachea. The tracheitis of this stage is a very characteristic condition, for one finds, in addition to the other signs affecting the nose, naso-pharynx, and larynx, that in the tracheal region there is audible without the aid of a stethoscope a loose rattling noise, produced by excessive watery secretion there, which induces at intervals a short, loose, rattling cough of feeble character. This cough only partially empties the trachea of the surplus secretion, the irritation apparently not being sufficient to induce a strong expulsive effort. Later comes bronchitis, starting either in the acute form and passing into the chronic, or being chronic from the outset, due to the gradual extension downwards of the inflammation commencing in the nostrils. This is no fancy picture, but one of which the different stages of development may be studied any day amongst the out-patients at a children's hospital.

In the case of acute nasal catarrh the important fact to bear in mind is that if this is not carefully treated and thoroughly cured it is extremely likely to become chronic. Under suitable surroundings, namely, an equable and not too hot room temperature, rest, simple diet, and saline aperients, an acute catarrh will probably run its normal course of a week or ten days quite satisfactorily, and without any local treatment. Chronic nasal catarrh may follow on an acute attack, or be due to dirt, or retained secretion, or cold, with which are not unfrequently associated a faulty diet and hereditary tendency. It may commence in the anterior nares and spread backwards, or commence in the posterior nares and spread forwards. It is manifested by a sniffling or snuffling breathing; a chronic discharge from the nose, muco-purulent or purulent, which is of an irritating character, and leads to superficial abrasions in the skin around the alæ nasi; a nasal tone of voice, and a tendency to mouth-breathing and to broken rest when the child is asleep, night terrors, etc. Complications may ensue from the spread of the inflammation to the eyes, the ears, and the larynx.

On examination the mucous membrane of the nose is seen to be swollen and reddened, the passages are narrowed, and hardened masses of pus or muco-pus are adherent to the walls. In the posterior nares, also, there is swelling and reddening of the mucous membrane, and an œdematous condition of the adenoid tissue. In all cases of this nature the glands, which are deeply situated under the angles of the jaws, will be found to be enlarged. Domestic treatment will probably have been employed before medical advice is sought. The infant or child will have been regarded as the victim of "a cold," and consequently he has been burdened with extra clothes by day, buried in blankets by night, and kept in over-heated rooms until the first time he goes out of doors he inevitably gets a chill. He will also have been considered delicate, and has therefore been stuffed with food, cod-liver oil, and iron until the appetite failed, the system became overloaded with matter it was unable to get rid of, and the congestion of the nasal mucous membrane was steadily aggravated.

The rational treatment of such cases will proceed on somewhat different lines. The body clothing should be warm, loose, and not such as to burden the infant or child. The sleeping and day rooms should be cool, well ventilated, and free from draughts. The child should be out in the fresh air for active or passive exercise as much

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during daylight as possible, save in damp or foggy weather, when the indoor atmosphere is probably preferable. His cot should stand clear of the walls, without curtains or closed sides, so as to allow of the play of fresh air all around. The mattress should not be too soft, and the pillow should be a firm hair one into which the head cannot sink. Soft, downy pillows are most unsatisfactory for children, as the head quickly excavates a hollow; sweating ensues, and the head is practically poulticed all night and kept in a state of constant hyperæmia. The diet will call for careful supervision. It ought to be simple, unstimulating fare, administered at regular intervals. In the case of infants under nine months of age nothing but the breast or cows' milk and barley water is required. For older children simple puddings, porridge, bread and milk, fish, and mutton are the standard foods. Beef tea or beef, meat juices or essences are often given by anxious parents in the most reckless manner, and to an extent which frequently aggravates the complaint. I have convinced myself that in these cases the effects of over-feeding, and of over-stimulating food, and of indigestible food are manifested not so much by the condition of the tongue as by that of the nasal mucous membrane. The young and healthy stomach will tolerate much before it breaks down, but meanwhile the blood is overloaded with more matter than it can get rid of, and chronic hyperæmia of the nasal mucous membrane may be one of the results. It is difficult to fix the exact amount of food which an individual child may require, but it is a very good rule to be guided by the child's appetite. If simple, plain fare be given, a child will not take too much, whereas if he be coaxed to eat more at every meal than his appetite suggests he will certainly suffer from over-eating. Head colds and attacks of acute gastric catarrh are frequently combined in young children, and both may be traced in many cases to errors of diet or to over-feeding. A constitutional tendency to such naso-pharyngeal catarrhs must also be recognised in many families, and this will undoubtedly be aggravated in the case of children who live in towns during the short, dark, and damp days of the winter months. Consequently a prolonged residence in a bright and bracing seaside or inland place will often be necessary for a complete cure in obstinate cases, and will be beneficial in all cases.

The local treatment of the nose must be attended to. The nostrils should be painted, or irrigated, or sprayed with a simple lotion such as the following:

℞ Sodii bicarbonatis,
Sodii biboratis,
Sodii chloridi . . . āā grs. xij.
Glycerini . . . . ♏xx.
Aquam . . . . ad ℥j.

Sig. One tablespoonful to be mixed with two tablespoonfuls of warm water.

This may be used two or three times a day, according to the amount of discharge, the number of hardened crusts present, and the extent of the obstruction. A little hazeline cream or weak boracic ointment may be introduced into and around the nostrils at night. As the nature of this treatment is sedative, it is important that the opposite result, namely, disturbance, be not induced by struggling on the part of the child. Violent spluttering and coughing may have the effect of driving the fluid up the Eustachian tubes, with consequent middle-ear disease, but gentleness and care ought to obviate this difficulty. If the child cannot be taught to allow irrigation to go on quietly, *i. e.* with the fluid passing up one nostril and down the other, under low-pressure syphonage, easy respiration being carried on all the time, it is better to use the spray or brush. As the great object of this local treatment is to secure the patency of the nasal passages, when that object has been secured the irrigation should not be used oftener than is necessary, namely, once daily, as the mucous membrane is easily excited to fresh inflammation. It is advisable also to teach these children as soon as possible to blow the nose, not with those violent blasts which some like to indulge in, but with sufficient force to drive out all accumulated secretions and keep the passages clear. Picking the nose is to be strictly forbidden. The patency of the nostrils having been once secured, it is to be further maintained by breathing exercises, the child for a few minutes morning and evening standing erect, and with firmly closed mouth inspiring and expiring deeply through the nose. Such measures, if persevered with, will not only relieve the active symptoms of nasal obstruction and prevent the complications which ensue, such as permanent adenoid hypertrophy, but will

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be found to aid materially in the healthy development of the child.

The stomach and bowels will probably also require attention. When a proper diet has been ordered, the following medicine, for a child of five years, will aid in restoring the appetite and the functions of the excretory organs :—

℞ Sodii salicylatis,
Sodii sulphocarbolatis . āā grs. v.
Tincturæ rhei . . . ♏xv.
Tincturæ zingiberis . . ♏iij.
Aquam ad . . . ʒj.

Sig. One teaspoonful three times daily after meals.

If chronic constipation be present, the following mixture may be given for a few weeks :—

℞ Tincturæ nucis vomicæ,
Tincturæ belladonnæ . . . āā ♏iij.
Extracti cascaræ sagradæ liquidi . ♏x—xv.
Extracti glycyrrhizæ liquidi . . ♏x.
Aquam ad . . . . . ʒj.

Sig. One teaspoonful thrice daily after meals.

The acute gastro-hepatic disturbances to which these children are liable will often be prevented by a dose of calomel every week or ten days.

℞ Calomelanos . grs. ij—iij.
Sodii bicarbonatis grs. iij.
Sacchari albi . grs. iij. Ft. pulv.

Sig. One powder at bedtime as ordered.

When this treatment has been employed for some time no further tonics will usually be required, but in certain cases there may be indications for the use of iron in small doses, or cod-liver oil. Surgical interference by cauterisation or excision of any swellings over the turbinate bones is not to be advised unless the obstruction is complete, and patency of the nostrils cannot be obtained by other means. This will seldom happen. Surgical measures may seriously interfere with the normal development of the nasal mucous membrane, and cause much harm in after life. Even a considerable degree of obstruction in early childhood is best treated by simple measures, as the normal expansion of the nasal structures will frequently bring about a natural cure.

2. *Syphilitic Rhinitis.*—This is the most severe orm of nasal catarrh to which infants are subject, and is unfortunately very common. One must not regard every child with "the snuffles" as the subject of hereditary syphilis, and a careful local and general examination must always be made. The nasal discharge in this disease is particularly profuse, irritating, purulent, and often blood-stained. The nostrils are more or less completely blocked, nasal breathing is rendered impossible, and continuous feeding from the breast or bottle is so much interfered with that rapid wasting may ensue. The inflammation tends to spread deeply, producing ulceration and destruction of the lining membranes of the nose, and even necrosis of the bones, while the skin around the alæ nasi becomes ulcerated so deeply that on recovery permanent scarring is left. Accompanying evidence of the specific nature of the disease will usually be present in the form of ulceration of the lips or mouth, anæmia, enlargement of the spleen, and a rash on the buttocks or other parts of the skin. The affection runs an acute and destructive course, and often threatens life itself from the complete obstruction of the nostrils, which prevents feeding. The age of onset, usually during the first month of life, renders the condition more serious. When the general disease is recovered from, local changes may remain in the nose. The ulceration of the mucous membrane and necrosis of the bones leads to a falling in of the bridge of the nose, which becomes permanent, and produces a varying amount of nasal obstruction throughout the patient's lifetime. Further, the extension of the disease from the nostrils into the naso-pharynx leads to further changes of an obstructive character there. Active treatment ought to be adopted as soon as the condition is recognised. An alkaline lotion similar to that given for simple catarrh is to be used several times a day, sufficiently often to prevent any blocking of the nose from inspissated muco-pus. In addition to the simple alkaline lotion, which is purely of a cleansing character, I believe it is of service to add to this lotion perchloride of mercury in quantity sufficient to make the fluid used of the strength of 1 to 2000. Another method I have sometimes used with advantage is, after using the alkaline lotion, to brush out the nostrils thoroughly with black wash diluted with three parts water. The local application of mercury seems in these cases to check the discharge and reduce the swelling more rapidly than the alkaline lotion alone. Constitutional treatment by mercury, careful dieting, and the rest of the treatment for congenital syphilis must of course be carried out at the same time.

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3. *Other Forms of Obstruction.*—(*a*) Foreign bodies, such as buttons, beads, small pieces of paper, etc., are not infrequently passed into the nostrils by children. Their treatment is of course easy when they are recognised, but as a matter of experience one finds that their presence is often overlooked. A child gives no history of the accident, but is simply brought for advice on account of a nasal discharge. In every case of discharge from one nostril only it is a good rule to examine carefully for the presence of a foreign body, both visually and with a probe. The nostril will usually be swollen, obstructed, and filled with purulent discharge. After a preliminary syringing the obstructing body can usually be seen, and can be removed by means of a bent probe or other instrument.

(*b*) Another form of obstruction is that due to fibrinous or membranous rhinitis. The symptoms in this affection are very like those of chronic catarrh. Nasal discharge, purulent or sanguineous, epistaxis, and nasal obstruction are present without any other local or general disturbances. On examination of the nostrils one finds that the mucous membrane is covered in whole or in part with a yellowish-white exudate, which is not merely inspissated muco-pus, but is membranous in character, and leaves a raw bleeding surface when detached. One or both of the nostrils may be affected with this membranous exudate, which has a tendency to re-form quickly after removal. The importance of this condition lies in the fact that it is really a form of localised diphtheria, and that virulent Loeffler's bacilli swarm in the membrane and the discharge. The infection, therefore, may spread either along the nostrils to the naso-pharynx and larynx of the patient, or to other members of the family from contact. At the same time it must be stated that as a matter of clinical experience the disease usually remains confined to the nostrils without signs of general infection, and without any great tendency to spread amongst children brought in contact with the patient. Dr. Lambert Lack,* who has made a study of this affection, concludes that it is an infectious disease with a special tendency to reproduce itself. He has never found a single case of unmistakable diphtheria traceable to a patient suffering from membranous rhinitis. The affection is a very chronic one, and may persist for several months. If the nasal obstruction is great, it is advisable to remove the membrane by means of forceps, an anæsthetic being employed if necessary, and possibly this may have to be done more than once. Irrigation of the nostrils with alkaline lotions (*v. s.*) should be employed two or three times a day, and with these may be alternated mild antiseptic lotions containing Condy's fluid, or carbolic acid, or corrosive sublimate. The evidence in favour of the necessity for complete isolation of such patients is by no means complete, but it is certainly advisable that a child thus affected should sleep alone, and have handkerchiefs, towels, etc., exclusively for its own use, and that these should be carefully disinfected afterwards.

(*c*) Hypertrophied mucous membranes, and (*d*) a deviated septum. These conditions when sufficiently pronounced to produce obstructive symptoms can only be relieved by surgical treatment, and this is seldom necessary in childhood.

* 'Fibrinous or Membranous Rhinitis,' London, 1899.

The Influence of Chronic Pulmonary Tuberculosis upon the Nerves and Senses.—H. Engel emphasises the influence of tubercular processes upon the nerves and senses, and the frequency of the so-called neurasthenic tuberculosis. It seems probable that the neurasthenic appearances are brought about primarily by the action of the tuberculous toxin upon the nerve-centres, although in many instances this action undoubtedly extends to the entire nervous system. The treatment of neurasthenic tuberculous patients demands the greatest care. In most instances they must be treated essentially as neurasthenics, and brought within the close personal control of the medical attendant. In such patients the administration of stimulants and narcotics must be carried on with the greatest caution. Neurasthenic stomach disturbances often give rise to the greatest apprehension, since they, as a rule, markedly interfere with the general nutrition. Usually such disturbances are the most successfully treated by lavage.—*Medical Record*, September 20th.

We have received from Burroughs, Wellcome, and Co. a sample of "Tabloid" lithium citrate and sodium sulphate (effervescent); when added to the requisite quantity of water it provides an effervescing draught of service in gouty and other conditions in which it is desired to administer lithium citrate with a gentle laxative.

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A CLINICAL LECTURE

ON

SOME CASES OF RAYNAUD'S DISEASE AND ITS ASSOCIATION WITH SCLERODERMIA.

Delivered at Westminster Hospital, June 3rd, 1902,

By W. A. WILLS, M.D., F.R.C.P.,

Physician to Out-patients, Westminster Hospital, and Senior Physician to the North-Eastern Hospital for Children, Hackney.

GENTLEMEN,—I have chosen as a subject for a clinical lecture to-day Raynaud's disease, including in my remarks certain allied conditions, such as frost-bite, chilblains, and sclerodermia. I happen to have had recently several cases among my out-patients, which illustrate some phases of these conditions, three of which I am able to show you to-day.

A person who is suffering from Raynaud's disease may be said to be abnormally sensitive to the effect of cold, of surface chill from cold weather or cold water, or chill produced by rapid evaporation from the body surface. In such people the result of such exposure is not mere temporary pallor or blueness of the exposed parts, but a more lasting pallor or blueness, which has a distinct tendency to recur periodically, and to be associated with various other symptoms shortly to be referred to.

Raynaud's disease is usually classified under three headings,—local syncope, local asphyxia, and symmetrical gangrene; and I would first point out the association between these three divisions and show you two cases to illustrate the stages.

Local syncope shows itself in "deadness" of the fingers or toes, which generally commences somewhat suddenly, and affects the corresponding fingers of the two hands. It may affect one finger only, or several fingers, or even the whole hand and wrist. If it comes on in one hand first, the corresponding part of the other hand almost immediately follows suit, and the successive affection of the various fingers follows the same order in each attack.

There is a complaint of pain, or at least of discomfort, in the affected parts, with numbness, tingling, or stiffness. And, indeed, there is actually some difficulty in the performance of the finer movements, and the patient may be unable to write, sew, or pick up small objects. No doubt this is due in part to the blunting of the tactile sense, which seems to be more affected than the sensibility to heat and cold.

The attacks last for very varying periods—a few minutes to some hours,—and are associated with some actual diminution in the flow through the arterioles of the part, though in many cases the radial pulse does not seem to be altered in the attack. If the pallid finger be pricked there will usually be no bleeding—a conclusive proof of the constriction of the arterioles.

As one might suppose, at the end of the attack there is a reaction, and the pallid fingers become red, sometimes even a little swollen, and are the seat of burning pain or tingling.

These attacks recur with regularity, though the interval varies in different people, and with such circumstances as exposure to cold or the occurrence of exhaustion.

There is, moreover, a general tendency for the attacks to increase in frequency over a certain period, then gradually to diminish in frequency until they finally cease. Such a cycle may last over some months.

Naturally sufferers are more prone to attacks during the colder months, but at all times any exceptional cold will almost certainly bring on an attack. The relationship of this affection to the "dead hands" or "dead fingers" which so many people suffer from after bathing in really cold water is very clearly seen, and the gradual transition from results which are merely physiological to those which are regarded as pathological emphasises the impossibility of defining accurately what is "disease."

If you want to see how common this condition of "dead hands" is, which must yet not be regarded as a diseased condition, you have only to visit a swimming-bath and look at the extremities of those boys who are spending some hours in and out of the water. They will almost all be found to be subject to this change; and a further significant fact has been observed under similar conditions, that the urine of nearly every one of them temporarily reacts to the test for albumen.

Local defects of circulation also have their influence in producing this condition, and I am acquainted with a man who received a severe injury to one finger, which divided the digital

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artery on one side of it; since which very slight exposure to cold produces deadness of this finger, which is only followed by deadness of the other fingers if the cold be more prolonged and severe.

While the stimulus which produces Raynaud's local syncope comes from without, yet any diversion of a large portion of the blood to a particular part of the body often brings on an attack, the best instance of which is the production of an attack by the eating of a hearty meal, when the digestive organs require a large extra blood-supply, and so lessen the amount of blood circulating in the extremities.

And though I am not aware that close mental application actually brings on attacks of local syncope, yet it seems not unlikely that it might, seeing how invariably in many people mental activity produces cold feet and even sometimes cold hands. Violent emotion has been recorded as the exciting cause of attacks, and it is significant of its nervous origin that the affection is more common among women than among men.

I had hoped to show you a case of Raynaud's disease of this type, who also came to my out-patients three weeks ago, but he has been prevented from coming. He is one of the "syncope" type, and has never had any asphyxia or gangrene.

His history is shortly as follows:

For the last five or six years his hands go cold and dead on exposure to cold. The attacks usually begin in the forefinger of the left hand, and spread to the third, fourth, and fifth fingers. The right hand then follows in the same order. He has never noticed it in the toes, nose, or ears, although his feet are rather cold at times. There has been no hæmoglobinuria.

He has the attacks daily even in summer, but they are less marked in the warm weather, and he then only has cold hands, which do not become white. He has several brothers and sisters, but only one has similar attacks in the left forefinger.

I obtained no history of syphilis.

Turning to the second form that this affection takes, that in which "local asphyxia" is the most prominent symptom, we find a very closely analogous set of symptoms. The difference between the first and second divisions of the affection appears to depend on the incidence of the vascular spasm: if this occur chiefly in the arterioles we get a diminished flow of blood to the part; if, on the other hand, the venous side of the peripheral circulation be affected, we get congestion of the part, owing to the interference with the escape of the blood into the larger veins. Sir Thomas Barlow quotes a case which seems to prove the existence of venous spasm. "In one case" which he observed "during the march of the paroxysm portions of the veins on the the back of the hand became quite moniliform; that is to say, there was an alternation of small dark swellings with narrow, almost colourless intervals between them. Whilst under observation the dark swellings gradually altered their position along the course of the veins, pointing to a varying contraction of the walls of these vessels."

I quote this observation because it states a definitely observed fact, thus differing from many merely conjectural statements which have been made about the mechanism of these paroxysms.

It shows the close relationship between the cases of syncope and those of asphyxia, that pallor of very short duration—it may be only momentary—often precedes the blueness; it looks, indeed, as if the spasm, similarly to that seen in the veins by Sir Thomas Barlow, commenced on the arterial side of the capillaries, travelled along them, and only became arrested for an appreciable period when it reached the veins.

To take a typical example of an asphyxial attack, the patient's fingers, after washing in cold water, become pale and white for a moment, then red, purplish blue, and finally almost black: this may occcur in one finger first, and then spread gradually to the others of the same hand, after which the other hand follows suit; or it may be that the index finger of the right hand alone is affected, and the same finger of the left hand alone suffers in sequence. The coloration then spreads to the hands, the wrists, and perhaps the forearms as far as the elbows, but probably not above. With this there will be pain in the affected part, often very severe, and of course the livid skin feels cold to the touch, while pressure leaves a white mark behind it for some considerable time; tactile sense may be impaired, and there may be analgesia, but heat and cold is still appreciated; stiffness of the fingers and inability to make finer movements are also present.

The duration of the whole attack may be from half an hour to many hours, and the attacks may

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be repeated as often as several times in one day, or they may recur at the same time (and probably from the same exciting cause) every day; the whole cycle thus being spread over several weeks, or even over as much as six months. Naturally these cycles are much more apt to occur through the winter months rather than the summer.

It may be mentioned that the nose and ears, the prominent parts of the cheeks, and even the buttocks are sometimes the seat of these changes.

The third main heading under which this disease is usually described is "symmetrical gangrene." This condition is really a sequel of one or both of the previous phases, a sequel that may occur quite early in the disease, or, on the other hand, be long delayed, and in some cases perhaps never reached.

It is a very natural result of such changes in the circulation as occur in this disease, and if the degree of local asphyxia be great, it would be more wonderful if some local necrosis did not occur.

So we find the gangrene affecting those parts where the local syncope or asphyxia is earliest found or most pronounced, namely, the tips of the fingers, toes, or nose, or the margin of the helix of the ear. The actual gangrene usually supervenes upon a severe attack or repeated attacks of the preceding condition, the skin of the fingers becoming gradually more tense and shining, and the finger more tapering until finally a small bulla appears, which breaks, collapses, and leaves a small black spot of exposed cuticle, which is in process of molecular death. The area is small, and the resulting scar and loss of tissue is correspondingly slight, but still repetitions of this localised gangrene do ultimately produce most serious changes in the affected fingers. For it is rarely one finger only that is affected, the condition is essentially multiple and symmetrical, and in the end the patient's hands are most seriously crippled.

One is at once struck by the similarity between this condition and frost-bite. In both you may have an attack of mere local syncope or cyanosis, which may pass off without any loss of tissue; in both the affection may be so severe as to produce a local necrosis. Indeed, the two conditions, apart from the recurrent nature of the one and the occasional and accidental nature of the other, are essentially similar.

It requires a particular habit of body for a slight chill to induce an attack of Raynaud's disease. It requires an exposure to very severe or prolonged cold to produce frost-bite, but out of a whole party of men or women exposed to such cold, only one or two will probably suffer from frost-bite. This has been seen over and over again in the history of arctic and alpine exploration; and while it is, no doubt, on the whole true that such cold-resisting power will be found to be possessed on the whole rather by the big and well-developed members of the party, there are sometimes striking exceptions to this rule, and some big strong men may find by experience that arctic exploration, for instance, is for them practically an impossibility.

Similarly, while it is usual for writers to point out that symmetrical gangrene is quite a different disease from chilblains, yet it always seems to me that the affinity is very close; people who suffer from chilblains suffer usually from other indications of a feeble peripheral circulation, such as cold hands and ears, blue hands, chapped hands, and the like. The onset of the chilblains is dependent on the season of the year, or on some special exposure to surface chill; they occur chiefly in the young, and often tend to improve with advancing age, and their site is always on the extremities,—in fact, their distribution coincides closely with that of the areas affected in Raynaud's disease.

It appears to me that the affinity between these three conditions, Raynaud's disease, frost-bite, and chilblains, is much greater than that between Raynaud's disease and peripheral neuritis, or even arteritis obliterans, which diseases have been suggested in some cases as being the fundamental basis of the associated symptoms to which the term Raynaud's disease has been applied.

A case that illustrates some of the foregoing points is a woman æt. 35, Sarah M—. She came up complaining of hemicrania, and is obviously of a neurotic temperament. I found her hands covered with chilblains, and, on further inquiry, elicited the fact that her hands had been cold and blue for the last two years, and that she has had chilblains in the winter. The attacks commence with numbness and whiteness of the left forefinger, and spread to the left third finger, but the right hand is not so affected at all, nor are her ears or nose.

After these attacks began two years ago she commenced to have chilblains on her fingers, which

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had never been the case previously, though she had had them on her feet since childhood. Her nails soon became affected in the manner they now are, being brittle, maculated, and uneven. There is, in fact, present in the matrices the same patchy vascular changes that mark the fingers and hands.

It is noticeable that her hands first became affected when she gave up her ironing work and took to packing meat essence tins. Obviously the warmer occupation prevented her hands from becoming chilled in the same way as her feet.

In her case there is no history of any other member of her family being affected similarly.

Let us further consider two other symptoms which are occasionally met with, the latter, indeed, somewhat frequently, temporary dimness of vision and hæmoglobinuria.

In several cases dimness of sight has occurred, which, on ophthalmoscopic examination, has proved to be due to narrowing of the central artery of the retina and its branches. But curiously enough this condition has in some cases alternated with the local asphyxia of the extremities, in others coincided with it. Certainly one can more easily understand the latter association, unless it be that the spasm progresses from the extremities to the retinal arteries in the same way that it spreads from the hands to the arms. But the parallel is not accurate, as the hands do not recover before the arms become affected, but merely anticipate them in onset and recovery, so that there is always in the attack a period when both are affected together.

Hæmoglobinuria, however, is a much more common symptom. I rather hesitate to use so definite a word as "symptom" when I find Dr. Monckton Copeman writing in Professor Allbutt's 'System of Medicine,' that "notwithstanding their frequent association, it may be doubted whether there is any relation between them other than that paroxysms of both are apt to be induced by the same cause, namely, exposure to cold;" but, on the other hand, the relationship between the two sets of symptoms is so close that I cannot help feeling that there must be more than a casual connection.

Still, it is perfectly clear that a considerable number of these patients do present this symptom, that the exciting cause, cold, in some patients results sometimes in asphyxia, sometimes in hæmoglobinuria, sometimes in both together. In a case recently shown at the Society for the Study of Diseases in Children by my colleague, Dr. James Taylor, which I had an opportunity of seeing on several occasions whilst the girl was in the ward at the North-Eastern Hospital for Children, the hæmoglobinuria was the only symptom at first noticed by the parents, and later some pallor of the fingers appeared. When she was admitted and carefully watched, it was seen that there was distinct but transient blueness of the fingers, which appeared soon after the child shivered, but before she passed any coloured urine. I happened to be in the ward when one of these attacks came on, and my attention was called to her hands by the Sister. The blueness was quite definite; but still so slight that one can easily understand its not being noticed by a careless observer. These attacks persisted through about three years, only occurring about once a fortnight if the child was kept warm, and less frequently, of course, in the summer; but latterly the urinary symptoms have entirely ceased, though the local asphyxia recurs from time to time.

This case is very interesting in another connection. It has been found that syphilis, usually congenital, but sometimes acquired, has been present in a considerable number of the recorded cases of Raynaud's disease. There was a distinct maternal history indicating syphilis in this child, but nothing about the child herself (aged 9) to suggest syphilis, until quite recently, since the disappearance of the hæmoglobinuria, when ulceration of the palate has supervened, and the uvula has been entirely destroyed. She also still suffers from oxaluria, which it has been found is a fairly common symptom in such patients.

One of the difficulties in associating Raynaud's disease and hæmoglobinuria appears to be that the changes which occur in the blood in the latter affection, namely, the rapid destruction of the red blood-cells on exposure to cold, have not been found generally in cases of Raynaud's disease, but some observations which have been made seem to show that similar changes occur in the blood when drawn from the affected fingers during an ordinary attack of local syncope and asphyxia. It has always been a question whether the blood changes found in hæmoglobinuria occurred in the peripheral circulation or in such central organs as the liver and spleen. Dr. A. T. Myers showed by his

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examination of the blood drawn from the finger in the case of a patient who suffered from Raynaud's disease with marked hæmoglobinuria, that in such a case destructive changes in the blood *were* present in the peripheral vessels. Putting these observations side by side, and taking into consideration the fact that unless the destruction of red blood-cells be very large the hæmoglobin may be dealt with by the liver and excreted in the form of bile pigment, rather than by the kidney in the form of hæmoglobin in the urine, it does not seem very difficult to associate the condition known as paroxysmal asphyxia and that known as paroxysmal hæmoglobinuria in a common origin—some undue sensibility of the organism to cold, whereby results are produced from the slightest chill which never occur in an ordinary person.

We may in this condition also consider the albuminuria to which young men are liable as a result of a surface chill, to which reference was made earlier in this lecture, for the class of cases to which the term cyclical albuminuria has been applied has a great affinity to this condition. There is a similar causation, a similar periodicity, and the affection occurs also mainly among the young.

Let us turn to the condition which the patient I am now going to show you mainly illustrates at the present time, Sclerodermia. She does not present this condition as completely as she did, as I am glad to say that under treatment she has considerably improved. Still I think it is obvious that there is much thickening and stiffness of the skin and subcutaneous tissue over the forearms, arms, neck, chest, and back, reaching down as far as the umbilicus. I cannot myself see that there is now any thickening of the skin of the face, which was the part first affected. The patient herself considers that it is now practically normal.

The thickened skin has a reddish appearance, and there are numerous white streaks of the type of lineæ albicantes. Round the waist there is a very distinct line of pigmentation, which has been more marked than it now is, and which reminds one greatly of the pigmentation of Addison's disease.

This skin condition answers exactly to the description of sclerodermia, between which and Raynaud's disease a distinct connection has been shown in several cases by Sir Thomas Barlow, Mr. Hutchinson, and others. It is true that in her case it began in the face, whereas the local asphyxia was most marked in the hands, but it early affected the hands and spread up the arms to the chest.

When at its worst it caused her considerable discomfort, not to say pain, but now she hardly feels it.

It is more common to find this peculiar skin change in a localised form in Raynaud's disease—especially affecting the fingers which have been asphyxiated. The thickening of the skin here soon gives place to atrophy, and the fingers become reduced in size, stiff, white, and tense, so that in course of time the hands may be almost useless. This atrophic condition of the fingers is naturally apt to be associated with local gangrene.

In the earlier stages of this affection recovery sometimes takes place, but when the "hide-bound" condition is fully established the prognosis is not good, and several patients have, in the course of a few years after its establishment, died of gradual exhaustion, wasting, and marasmus. The outlook is certainly unfavourable when once the atrophy of the skin has occurred.

Eliza T—, æt. 50 years, a dressmaker. She always suffered from cold hands and feet since a child, but never noticed any whiteness or blueness of the hands until two years ago. The hands had been going white for about a year before they went blue. The feet became white about the same date as the hands. In the attacks the right forefinger was first affected, then the third, fourth, fifth, and last the thumb, but the syncope never extended beyond the knuckles. The left hand followed the right. The feet follow the hands, but patient does not know which foot is first affected.

The attacks of local syncope, after lasting a year, were succeeded by similar attacks of local asphyxia, the parts being affected in the same order, but the blueness extending farther than the whiteness—up to the wrists. In this second stage there was no whiteness preceding the blueness. The feet, however, did not become blue until about six months ago when she was in hospital.

There was stiffness of the fingers, with inability to pick up small objects during the attacks, no loss of sensibility to pain, but impairment of temperature sensation.

While in the hospital she had attacks of localised

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perspiration on the arms and chest after the electric baths.

She felt tingling in the hands as they were recovering, but no severe pain. The attacks were not brought on by eating, as is sometimes the case.

Her nose and ears were at no time affected, and she never had any chilblains nor any other evidence of local gangrene.

If nothing was done the attacks usually lasted about three or four hours, but she generally put her hands into warm water, which caused the attack to pass off. She had two or three attacks a day, as the slightest exposure to chill, such as passing from a warm to a cold room, brought them on.

She has had pain in shoulders and elbows, but never any definite effusion, and no pain in the knees or ankles.

There seems to have been a continuous increase in the severity of the symptoms from the onset to the development of asphyxia, and then a few months later a gradual improvement.*

She first noticed the thickening of the skin about June, 1901, beginning in the face and then on the forearms, arms, shoulders, and chest as far down as the waist, and perhaps over the lower part of the abdomen, but never on the thighs, legs, or feet. A line of pigmentation round the waist appeared about the same time. The tightness of the skin was accompanied by a peculiar feeling of pressure, and this produced a feeling of difficulty in breathing.

There is no evidence of any hæmoglobinuria at any time, though the urine was high-coloured about a year ago.

She has noticed some dimness of sight during the last few months, but not when she had the most severe asphyxia, during which period her sight was good. The attacks of dimness of sight come on when at work, and last about a quarter of an hour, going away after resting.

She has had no previous illness except diphtheria, when aged seventeen, no ague, and there is no evidence of syphilis.

She has five brothers, three of whom have died of phthisis; the two living brothers and one sister have no sign of Raynaud's disease, but one of the brothers who died used to have white fingers and suffer from cold. Father and mother both alive, and have never had any such symptoms.

The third patient is a somewhat different case, and I cannot class her as a case of Raynaud; and yet the circulatory changes in her fingers obviously present an analogy to this disease.

She came to me as a case of rheumatism, having suffered from pain, swelling, and stiffness of the fingers for some five or six years. And though there is swelling of her fingers, there is no swelling of her joints, and the joint movement is free, except for its limitation by the swelling. Her hands also were always cold, and she had a difficulty in performing fine movements.

With the X rays the bones and joints can be seen to be quite normal. This condition is obviously due to some circulatory disturbance of the same type as that found in Raynaud's disease, only in Raynaud it is paroxysmal, and here it is continued. The condition has an analogy to that described as erythromelalgia, and perhaps to angioneurotic œdema, but it differs too much from these to be so classified. It really perhaps corresponds more closely to an early stage of sclerodermia, of which you have already seen an example.

It is noticeable that she has derived great benefit, even if it be at present rather transitory, from galvanism applied in the form of a hand-bath. Immediately after the application her hands feel quite warm and supple.

The question of treatment now remains to be considered. Naturally, in a person who has a certain temperament of body, one cannot expect to produce any profound change by ordinary drug treatment. So one is not surprised to find that drug treatment is not very satisfactory. Undoubtedly we often find that such patients are anæmic, thin, and by no means robust, and in such cases the ordinary treatment by tonics and good feeding is indicated. But it is no uncommon thing for such patients to say that their general health is excellent. Therefore one soon finds that the question of local treatment has to be considered, especially in the case of those who cannot take advantage of warm climates during the winter. For undoubtedly it will be a great advantage for such patients to spend their time in perpetual summer, and this they may

* I have recently (September) seen this patient. With the onset of colder weather her symptoms have all returned, including the feeling of tightness from the sclerodermia on the neck and chest.

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do by living in the Northern Hemisphere in our summer, and the Southern Hemisphere during our winter (which is, of course, their summer); but such climatic advantages must necessarily be reserved for the well-to-do persons only.

One patient of mine, whose affection was chiefly of the chilblain type, although there was also distinct local asphyxia from time to time, went out about eighteen months ago to the Cape. Of course this was not an ideal climate for a case of Raynaud's disease, as the South African winters, though bright, sunny, and short, are very cold. I heard from her after she had been out there about a year, and she said that the summer months suited her admirably, and that her hands and feet got almost entirely well. Her description of the effect of cold is very striking. "I do feel so different in hot weather, quite happy and well; I often notice, if it has been very hot, that as the sun goes, I begin to get cold, and my hands and feet turn blue." This shows well the immediate effect of surface chill.

To return to the question of local treatment, patients soon find out for themselves that the immersion of the hands and feet in warm water will often cut short an attack; but this is merely palliative, and probably only renders them more sensitive. In the same way they learn how necessary it is to keep their hands warmly covered with gloves, and use a muff in cold weather. Friction, massage, the use of stimulating liniments may be tried, but the skin is easily injured owing to its impaired nutrition, and such applications are not very satisfactory. Where there is any local necrosis of the skin it is impossible.

Hence one soon finds that it is necessary to have recourse to electrical treatment, which is of undoubted use, and not only alleviates but probably actually wards off further attacks. To be of any use, it requires to be carried out for some little time, some months perhaps, daily, or at least three or four times a week.

The galvanic current should be used, and best in the form of a hand or arm bath. Either one pole of the battery should be attached to the upper part of the limb, while the other pole is placed in the basin in which the extremity (hand or foot) is immersed, or both poles may be immersed in the basin—which must be earthenware and non-conducting—and placed as far apart as the vessel allows. The hand is held in the warm water or warm salt and water, and the current gradually switched on, until its strength is such that the patient cannot bear more. This must be maintained for ten, fifteen, or twenty minutes. The pressure will probably be some forty volts, and the quantity registered by the ampère meter some thirty-five milliampères.

While the hands are immersed the fingers should be moved about by the patient. When the hands are withdrawn it will be found that they feel warm, that the skin is red and tingles, and the movements of the fingers are more easily performed. This condition persists for some time, even some hours, after the application.

If there be chilblains or local patches of gangrene it will be found that these soon begin to heal and recover. In fact, for a certain number of hours in the day the nutrition of the limb is much improved.

No doubt, in the case of patients who can afford it, an electrical installation of their own, preferably such as will enable them to use the electric lighting current in their house is the best, as they will then be able to use the current once or even twice a day, and especially to ward off any attack as soon as it threatens.

I know of no other form of treatment which will give so much relief, and tend so much to produce permanent improvement.

Treatment of Bubo.—Cedercreutz says that in at least one seventh of all cases of inguinal buboes the venereal cause cannot be established. They should be treated as far as possible by conservative expectant measures, especially bags of hot sand, alcohol bandages over zinc paste, dressings with aluminum acetate, and in the very earliest stages, ice-bags. When suppuration has set in, and especially in case a single gland is involved, a small incision followed by one or more injections of a 1 or 2 per cent. solution of silver nitrate, or better still in most cases of 10 per cent. iodoformed vaseline, will be found effective. About 75 to 80 per cent. of the cases treated on this plan heal without necessity for extensive operative intervention. Curetting and tamponing with iodoform gauze is the last resort, or total excision of the gland.—*Journ. A.M.A.*, September 27th.

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A SUMMARY OF THREE CLINICAL LECTURES ON THE NATURE AND ÆTIOLOGY OF PERNICIOUS ANÆMIA, BASED ON TWENTY-FIVE CASES.

Delivered at the Medical Graduates' College and Polyclinic, May 1902.

By WILLIAM HUNTER, M.D., F.R.C.P.,

Physician to the London Fever Hospital; Examiner in Medicine, University of Glasgow.

LECTURE I.

GENTLEMEN,—My account to-day is on the nature and ætiology of pernicious anæmia, based upon new clinical and pathological observations made in twenty-five cases which have come under my notice during the last two years, these investigations being additional to those recently recorded (1900).

GENERAL ÆTIOLOGY.—The facts in connection with the general ætiology in the twenty-five cases may be summarised as follows:—The *age* varied from thirty-two—the youngest, to seventy-two—the oldest. The average age was fifty-one, the majority of the cases being between the ages of forty and sixty. With regard to *sex*, nineteen cases were in men and six in women. As to *station in life*, there were seventeen private patients and eight hospital patients. The previous history in all the cases was good. In no case did *poverty* play any part in the production of the disease; nor did *bad food*, nor *insufficient food*. As one of the hospital patients put it, he had always been careful about what he ate and drank, and had been in a position to gratify his wishes in those respects. In none of the six cases in women did *pregnancy* or the *puerperal troubles* play any part. In no case was there any previous *loss of blood*, and in the few cases—the very small minority—in which epistaxis occurred, this was only slight in amount and not

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very frequent in occurrence; it occurred only after the disease was already well developed, and was not a factor in its original production. In none of the twenty-five cases was there reasonable ground for suspecting *malignant disease* (two cases in which this question arose I have purposely excluded), and in none of the seven cases examined post mortem was any malignant disease found. A history of antecedent *gastric* or *intestinal disturbance*, as I shall presently show, was found in every case. In no case was this of sufficient character or intensity to account for the supervention of such an intense degree of anæmia. The various disturbances connected with this tract have an important significance, which I shall bring out, not as causes of the disease, but as symptoms of the gastric and intestinal infective lesions which I find to be associated with the disease.

In all the cases observed during life, twenty-five in number, and in the cases, seven in number, in which post-mortem examinations were obtained, I found definite *lesions* in the *mouth*, *tongue*, *stomach*, or *intestine*—either singly or associated with each other. These were (1) a special form of glossitis (in every case); (2) preceding or existing "oral sepsis" (twenty-four cases); (3) "septic gastritis," the new condition I have recently described as a common result of long-continued oral sepsis, causing finally gastric atrophy (more or less in every case, and most intense in three cases); and (4) croupous enteritis, congestion and catarrh of intestine, follicular enlargement in intestine, and slight patchy colitis (three cases). With regard to these, the point I wish to emphasise to you now, in view of the significance which I shall subsequently attach to them, is this:—In no case were these lesions, either in character or in degree, sufficient of themselves to account for the intense anæmia by any interference with nutrition that they may have been capable of producing. Lastly, in connection with the important part I have found reason to ascribe to sepsis as one of the factors concerned in producing this disease, I would draw your attention to the subject of various *chronic discharges* and *suppurations*. In no case were there any such discharges or suppurations sufficient of themselves to cause this anæmia.

To sum up, then, in all the twenty-five cases observed, the anæmia might well have been described in Addison's own words—"occurring without any recognisable cause whatever; cases where there has been no previous loss of blood, no exhausting diarrhœa, no chlorosis, no purpura, no renal, splenic, miasmatic, glandular, strumous, or malignant disease." On the other hand, in not one of the cases could Biermer's description of the ætiology of "progressive pernicious anæmia," which has so largely influenced the views of most subsequent observers, be applied.* His statement was that "progressive pernicious anæmia was found chiefly amongst poor people, especially among women, among whom, in addition to poverty, puerperal conditions appeared to be the favouring cause. Insufficient and unsuitable feeding, unhealthy surroundings, chronic discharges, especially persistent diarrhœa, sometimes also hæmorrhages, usually preceded the disease and caused it. The most common cause, according to his observations, was chronic diarrhœa, with or without gastric disturbance. A spontaneous origin without clear ætiology was the exception." So, gentlemen, the first clear conclusion in regard to the ætiology I have to draw your attention to is this: that even *on clinical grounds* this disease is not one which can reasonably be ascribed to the ordinary causes of anæmia, since (1) it occurs without the presence of these, and (2) these are often present in severest degree without causing the anæmia. In that respect Addison's original ætiological foundation has been proved, as far as my own work is concerned, to have been much more solidly laid than that of Biermer. And this conclusion holds, if possible, still more true *on pathological grounds*, since none of these conditions, however severe, can produce the pigment changes in liver, kidney, and spleen characteristic of pernicious anæmia. The following analyses in eleven cases recently observed show the large excess of iron in pernicious anæmia, as contrasted with the percentage in the anæmia of profound sepsis and malignant disease.

Analyses.

Percentage of Iron.

Pernicious Anæmia.

	Liver.	Kidney.	Spleen.	
Case 1 ...	0·360 ...	0·033 ...	0·069 ...	1900
Case 2 ...	0·240 ...	0·042 ...	0·094 ...	1900
Case 3 ...	0·363 ...	0·133 ...	0·141 ...	1900

* On this point, see 'Pernicious Anæmia,' and 'Brit. Med. Journ.,' May, 1902.

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Pernicious Anæmia (continued).

	Liver.	Kidney.	Spleen.	
Case 4 ...	0·114 ...	0·055 ...	0·111 ...	1901
Case 5 ...	0·292 ...	0·013 ...	0·275 ...	1901
Case 6 ...	0·140 ...	0·256 ...	0·099 ...	1902
Case 7 ...	0·385 ...	0·031 ...	0·090 ...	1902
Average ...	0·270 ...	0·090		
Total ...	0·360 ...		0·125	

Result.—A more than fourfold increase of iron percentage in liver and kidney. In all cases the percentage in *liver* alone exceeded that in spleen.

Normal.

	Liver.	Kidney.	Spleen.
Case 8 ...	0·080 ...	0·004 ...	0·090
Total ...	0·084		

Septico-pyæmia.

Case 9 ...	0·069 ...	0·014 ...	0·105
Total ...	0·083		

Cancer.

Case 10 ...	0·120 ...	0·022 ...	1·330
Case 11 ...	0·022 ...	0·003 ...	0·013
Total ...	0·083		0·671

Result.—In all cases the average percentage in spleen exceeded that in liver and kidney combined. In only one case (No. 11) was it less, but in that case the percentage in liver was only one fourth the normal.

Lecture II.

Infective Ætiology of the Disease.—Having thus made clear that ordinary causes of anæmia, *including sepsis*, however severe, cannot produce this anæmia with its characteristic pigment changes, I pass on now to consider another class of facts which appear to me to afford a much deeper insight into the ætiology of this disease. They denote, as I conclude, that this anæmia is not the outcome of malnutrition, or malassimilation, or abnormal fermentation processes in the gastric or intestinal contents—to each and all of which it is by most observers still ascribed; but that it is a chronic infective disease, caused by an infection of certain parts of the mucosa of the digestive tract, especially the tongue and the stomach, but occasionally also the intestine. These infective processes explain in my opinion many of the symptoms of the disease, including especially the various gastro-intestinal disturbances so often met, as also the fever; they also explain and account for the various lesions met with and described from time to time in connection with this tract, especially the atrophy of the gastric and intestinal glands, and lesions of a more definite character, congestions, erosions, ulcers, etc., occasionally described. These various lesions and the definite bacteriological evidence I have recently obtained of their infective nature I now demonstrate to you.

The infective factors which I find to be concerned in the ætiology of this form of anæmia are two in number: 1, a septic infection; 2, a specific (hæmolytic) infection. The evidence on which my conclusions regarding septic infection have been based has been of three kinds. (*a*) The prevalence of septic conditions—stomatitis and gingivitis, and other septic troubles in connection with the mouth *preceding* the onset of the disease—conditions which I have designated by a shorter title, "oral sepsis" ("Oral Sepsis as a Cause of Disease," 'Brit. Med. Journ.,' 1900, and 'Clinical Journ.,' 1900). These conditions have either been altogether overlooked, or been regarded as secondary to the anæmia. (*b*) The existence of a septic condition of the stomach not before described, viz. a subacute infective gastritis as a common result of long-continued oral sepsis—a real septic catarrh which I have designated "septic gastritis," set up by the continued influx of pus organisms into the stomach. (*c*) The occasional presence of similar infective lesions in the mucosa of certain parts of the intestinal canal, probably having a similar origin—"septic enteritis." These lesions (which I now demonstrate to you) by themselves, however severe they may be, are not sufficient to cause pernicious anæmia—an important fact I emphasise once more. But they constitute, in my judgment, a most important ætiological factor in the development of the disease. They create lesions of the mucosa in the alimentary canal favourable to any subsequent infection to which the tract may be exposed. Evidence of such subsequent infection is, I consider, to be found in (1) the onset of a special degree of *hæmolysis* (see Analyses), accompanied by (2) a *form of glossitis* in every case that has come under my observation from 1888 onwards. This glossitis, so obscure, and apparently of so little consequence that in the records of 250 cases there are only some twenty references to anything being

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wrong with the tongue, has hitherto been regarded, like other conditions of the tongue, as merely symptomatic of stomach disorder. In my experience, however, it presents such characters in respect of mode of onset, relation to onset of the anæmia, clinical features, and, as observed during life, pathological features as well, that I was led (1900) to the conclusion that we have in it to do not with an ordinary superficial glossitis, but with "a glossitis of a special character, depending on some deep-seated infection of the tongue itself." This conclusion is confirmed by the observations now to be recorded.

In connection with the ætiology of this form of anæmia there is, in short, evidence of a double infection: (1) a *specific* (*hæmolytic*) *infection*, of which the chief evidence is an *infective glossitis* presenting certain special characters; and (2) a *septic infection* of mouth, stomach, and intestine, of which the chief evidences are—*during life* varying degrees of "oral sepsis" and "septic gastritis" (the latter recognisable during life in the vomit), and various symptoms of oral, gastric, and intestinal disturbance; and *after death* the conditions of gastritis, gastric atrophy, intestinal atrophy, erosions, or ulcers now and again found in the disease. In the next lecture I propose to deal with the features of this glossitis, but before leaving the point of the septic factor I would draw attention to the further evidence obtained as to the existence of this factor in connection with the disease. Although this sepsis is not itself the cause of pernicious anæmia, it is an important factor in creating the conditions of the mucosa in the mouth and stomach and intestines which favour the actual onset of the special infection underlying the disease. In the further investigations made in the last twelve months I have been able to make bacteriological observations in three cases of this disease. In Case 1 the *Streptococcus longus* infection in the spleen; marked lesions in the tongue, but no bacteriological investigation made. Case 2, there was general streptococcal infection, very marked in the spleen and in the blood, and, as I shall show you presently, in the tongue. Case 3, very marked streptococcal infection in the tongue, yielding in pure culture the *Streptococcus longus*. In these three cases, then, there has been obtained bacteriological corroboration of the conclusion that associated with this disease there is a septic infection factor.

Lecture III.

Infective glossitis.—To-day I draw your attention to the other group of infective lesions, which are not common to other diseases, but which are, so far as my observations at present go, peculiar to pernicious anæmia, namely, a *glossitis*—and to the evidence which I have found of its being of an *infective* character. I wish to give you some statistics with regard to the nature of the further investigation now made. The account is based on (1) a post-mortem examination in seven cases of pernicious anæmia; with (2) histological and chemical examination of organs from these cases; controlled by (3) observations on some ten other conditions, namely, septico-pyæmia, health, septicæmia, cirrhosis of the liver, emphysema, mitral disease, cancer of stomach, cancer of tongue, tubercular laryngitis, and gastric ulcer. Eight hundred and eighty sections, chiefly of tongue and stomach, from the cases of pernicious anæmia, have been examined, and controlled by examination of 270 sections from other conditions, making a total of about 1150 sections; and (4) bacteriological observation on the tongue and vomit.

Clinical Features of the Glossitis.—The glossitis of which I now show you a specimen was from a case of pernicious anæmia, in which there was recurrent inflammation of the tongue. (P.M., *Streptococcus longus* isolated from spleen.) This recurrent inflammation is marked by great tenderness, soreness, and the appearance of various changes in the mucosa. The commonest change is the formation of little vesicles on the tongue, which are often accompanied by desquamation of the epithelium, so that there are small raw ulcers. At times the whole tongue swells up, so as to practically fill the mouth. These attacks recur at intervals of two or three weeks, and they have a very interesting relation to the onset of the anæmia. In every case the tenderness of the tongue is noticed on or about the onset of the anæmia, and is generally worse early in the disease. It tends to subside as the disease goes on, and in the later stages, when the patient is profoundly anæmic, the usual appearance presented by the tongue is that of a pale, clean, soft, absolutely unsuspicious-looking organ. If such a patient, however, be observed over a long period of time there is invariably, in all the cases which I have had under my care, a recurrence from time to time of the

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lesions, and of the symptoms connected with the tongue. So that the patient's periods of illness are associated with the recrudescence of tenderness and inflammatory changes in connection with the tongue, the inflammatory character of the lesions lessening as the disease advances, owing to the increasing degree of anæmia. The final changes which this lesion causes in the tongue are atrophy of the mucosa, so that the tongue becomes smooth and glossy—a condition almost resembling that in the disease known as psilosis, but presenting distinctive points of difference.

Pathological Characters of the Glossitis.—I show you now the histological lesions connected with the above changes in the tongue. First of all I show you a slide of a normal tongue, with an ordinary septic ulcer on the side. That is not the lesion of pernicious anæmia. And in no case of sepsis, however profound—and I show you a specimen from a patient who died with a foul oral sepsis which had caused pyæmia*—there are none of the changes in the tongue which I find in connection with pernicious anæmia. Next I show you a tongue from a case of *cancer of the stomach.* It shows a wrinkling, but the whole of this surface is covered by a perfectly velvety mucosa. If you compare the naked-eye appearance presented by this tongue with that of pernicious anæmia, there are marked differences. That tongue of cancer was from a case of profound anæmia, almost of the same age and the same degree of weakness and emaciation as the case of pernicious anæmia which I now show you. Contrast with these the *tongue of pernicious anæmia* now shown. Case 1: Three fourths of the entire surface is a smooth surface; it was glossy in life, and was deprived of its papillæ. On the other side there is a patch of mucosa left amidst the smooth surface, and the portion in front is fissured and cracked very markedly, as you will see in the specimen I show you, and it presents a very curious sodden and chronically inflamed appearance. Case 2: In the second case now shown the patient had a tongue which was from time to time tender, but throughout her life there was nothing strikingly wrong with the appearance of the tongue owing to the great pallor of the mucosa, except that from time to time it became tender, and a little red at points. Post mortem, however (the natural colours of the tongue having been preserved), you are able to see what a chronically sodden inflamed condition the mucosa of the tongue presents around the edges, around the dorsum, and over three quarters of its extent. This condition is exceedingly marked, and even in the photograph you can see how the surface is raised above the level. The contrast between this sodden, white appearance and the paleness of the dorsum of the tongue is exceedingly marked.

Histological Changes.—The histological lesions connected with these changes are still more striking. Here is the *normal tongue*, showing the thickness of the epithelium covering,—you see it occupies a very large portion of the field. The next is a section from the same tongue at a different part, where the epithelium is thinner in character. Notice the great depth of the epithelium, the thickness of the submucous tissue, and the muscular coat. The depth of the epithelium extends often to a layer of forty, sixty, or a hundred epithelial cells. Here is a specimen from the same tongue low down, where the mucosa is thin, and where the epithelium extends fifteen or twenty layers of cells. The mucosa is of moderate thickness. Now I show you a tongue from a case of *cancer of the stomach.* Notice in this that the epithelium is very well marked, and the papillæ also are very well marked. It is somewhat thinner than in the normal owing to the slight degree of wasting, but there is still a fair depth of epithelium, made up, as you see in the next slide, of well-preserved epithelial cells of the tongue. Contrast with these the slide now shown from a *tongue of pernicious anæmia.* You will notice in this that for a considerable distance there is an area denuded of epithelium, and covered with projecting granulations. The epithelium is retained at certain parts, but it is exceedingly thin and in parts ulcerated, the submucosa being in direct connection with the cavity of the mouth. The next is a high power presentation of the same section, showing the absence of epithelium from these granulations. In the next specimen, from another case of pernicious anæmia, another change is seen—a deep fissure, with intense inflammation. A large piece of tongue is projecting in an ulcerating condition, and, as you will presently see, in this specimen there was the most intense infection throughout the whole of this mass of tissue. In

* See Analysis, Case 9.

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the third case of pernicious anæmia now shown the changes are still further advanced. The epithelium is reduced to two or three layers of cells in thickness; indeed, it is a mere line, and at one part the epithelium is absent, so that the whole of the lymphatics in the submucosa are in direct communication with the mouth. The epithelium is reduced to two or three cells in thickness, instead of occupying the whole field as in the normal tongue. Here is another section from the same case, where the epithelium is reduced to a mere line on the surface of the tongue, and the greater part of the tongue is hollowed out to form large lymphatic spaces surrounded by atrophied muscular tissue. This is better brought out in the next specimen, where you will see there is no trace of epithelium and no trace of submucosa, and hardly any trace of muscular tissue; the whole thing is a wide lymphatic sac, the substance consisting of widely dilated lymphatics. The next is another specimen from the same case, and in order to bring out the degree of change I may say this is from an identical spot corresponding to the one where the epithelium of the normal tongue was of great thickness. You will notice here absence or extreme thinning of the epithelial layer, and the greater part of the tongue consisting of widely distended lymphatics. *These changes are of a kind and degree peculiar to pernicious anæmia*, and I have not found them in any of the control observations which I have made in all sorts of morbid conditions. Here I show you the actual changes under a high power, by which you will see that the whole mucosa is reduced to a thin layer of cells, and the epithelium itself is of a very atrophied and shrunken character. The widely distended lymphatics are well seen underneath. The changes in the epithelium which accompany these remarkable lesions in the tongue are very marked, namely, great swelling of the epithelial cells with mucoid degeneration, and in some parts, as the next slide shows, there are marked inflammatory changes in the submucosa.

Bacteriology of this Glossitis.—In connection with these changes there is a marked coccal infection. And now I come to a very interesting point, to which I wish to draw your attention. In the last two cases I have made bacteriological observations on the tongue, taking inoculations directly from the *substance* of the tongue with the minutest bacteriological precautions. In both cases I obtained *pure cultures of the* Streptococcus longus *unaccompanied by any other organism* cultures which I now show you. In both cases the patients had been under strict antiseptic treatment of the mouth for several months before their death.

Summary.—I have now to very briefly summarise the conclusions which appear to me to be deducible from these various classes of facts. The ætiology of this disease appears to me to be of this kind, that it is essentially connected with infective processes. These infective processes are of two kinds. The first is a septic process *preceding* the onset of the disease as the clinical evidence shows, and *existing along* with the disease as the bacteriological evidence now adduced shows. This is the septic factor connected with the disease, and its lesions and evidences are found in the mouth, stomach, and intestine,—that is to say, "oral sepsis," "septic gastritis," and "septic enteritis." *These lesions alone are not sufficient, and septic infection alone is not sufficient to cause the disease.* But in pernicious anæmia there is evidence of another infective inflammation, viz. in connection with the tongue. The changes which I have found in the tongue are of a definite character in association with pernicious anæmia, and in my judgment different from those which I have seen in any other disease. The history of the disease in its origin is generally as follows: an antecedent history of oral, gastric, or intestinal sepsis, the clinical evidence of which is the presence of various oral, gastric, and intestinal symptoms. All the patients in these cases described, twenty-five in number, have had an antecedent history of gastric or intestinal disturbance—nausea, sickness, indigestion, sometimes with slight diarrhœa or looseness of the bowels; and there has been a history of oral sepsis in all the cases with one doubtful exception. These may exist for some years without causing the disease. Suddenly on to this sepsis there is grafted a most intense anæmia, marked by extreme hæmolytic changes in the blood and urine. These denote the supervention of a new factor marked *pathologically* by intense hæmolysis, *clinically* by the occurrence of symptoms of tenderness and trouble connected with the tongue. This glossitis is of a peculiar recurring and persistent character, and the observa-

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tions I have made appear to denote that we have in it to do not with a mere symptomatic inflammation of the tongue, but with an actual *infective inflammation presenting special pathological features*. So that, of the features and changes which I have described to you, this peculiar glossitis is the one which I regard as most interesting from the point of view of the special nature of this disease. But the disease is not caused by the mere inflammation in the tongue or by the infection of the tongue; but the infection having once got root in the tongue, it is carried down to other parts of the mucosa, and affects the gastric mucosa in the great majority of cases, and sometimes also the intestinal mucosa. And this infection is permitted in these regions by the existence beforehand of the conditions of septic gastritis and septic enteritis which I have described. So that this disease presents itself to me as a definite infective disease, with a definite mode of origin, and with a definite antecedent history. It is a definite infective disease localised to the mucosa of the mouth, and of the tongue and stomach and intestines, causing changes which heal up in one part and spread to another, but always recurring and of great persistency. This infection is one which I regard as having been rooted in or associated with an antecedent condition of sepsis. The septic factor favours the contraction of the special (hæmolytic) infection. The source of this latter infection I have traced in five cases to a definite exposure to drain poisons. The various disturbances connected with the gastro-intestinal tract to which I drew your attention in the first lecture as having been considered to be causes of the disease are, in my judgment, not the causes but merely the symptoms of the infective processes in connection with the mouth, stomach, and intestine. They stand to the disease in the same relation as diarrhœa stands to typhoid fever. The diarrhœa in typhoid fever is not the cause of the typhoid fever, and the diarrhœa or gastro-intestinal disturbance in pernicious anæmia, however marked, is not the cause of pernicious anæmia; it is merely one of the symptoms associated with the lesions in this portion of the gastro-intestinal tract.

In concluding this survey of the nature and ætiology of pernicious anæmia I have led you somewhat into details regarding the nature of the disease. That happens to be the result of the somewhat detailed investigations which I have carried on, and which appear to throw some light on its nature. But if I were to briefly state the outcome of these investigations it would be to say this: that in connection with the nature, prognosis, and treatment of the disease it directs our attention to a new class of facts. As regards the *nature*, instead of this disease being an anæmia pure and simple, "without any particular symptoms, without any lesions other than those of the anæmia itself," as it has been previously described, this form of anæmia, in my experience, is a well-characterised infective disease. It may not always present the whole of its characteristic features at any moment while under observation, but the characteristic features of the disease are invariably presented if the patient be sufficiently long under observation. For example, it is not uncommon to have a patient lying for a month or two in the hospital under observation, with hardly any active symptom of any kind,—he is like a phthisical patient who has no sputum and no fever, and no physical signs in the chest. Later on, at another period, he presents an aggravation of all the features of the disease—increased anæmia, profound hæmolytic changes in the urine, glossitis, gastric and intestinal trouble and fever, and other nervous disturbances. These subside and become quiescent, later on becoming very profound. In connection also with *prognosis* these facts are of importance, in so far as they draw attention to the really important factors in connection with the disease, namely, the infective factors. It will remain for experience with later cases, and a larger number of them, to show how far these new facts will help us to combat the disease. My own hope is that the disease will prove to be more amenable to *treatment* than has hitherto been the case, and for this reason: that in my own experience it can be diagnosed at a much earlier stage than is often thought possible. It is easy enough to diagnose the disease when the patient is in a state of profound anæmia; but in all cases the course of the disease has been much longer, generally for a year or two preceding. By earlier diagnosis it may be possible to get rid of the infective factors which appear to me to be the chief ones in connection with the disease, and arrest the disease before it has by its long duration exhausted all the recuperative powers the patient originally possessed.

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ON THE RECOGNITION AND MANAGEMENT OF INTESTINAL OBSTRUCTION.*

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I AM sure you will allow that time cannot be wasted when it is spent upon this difficult subject. Had the notice been longer I might have introduced this discussion in a more worthy manner. But now I can only hope to tell you briefly some of my experience, and put before you some conclusions at which I have arrived. One might well have chosen a happier topic. There would be some gratification in telling of large numbers of successful appendectomies, ovariotomies, or radical cures. But such narrations are singularly wanting in instruction, and not always a compliment to the intelligence of the audience. As a matter of fact, my own experience of the treatment of intestinal obstruction has been most unsatisfactory, and I ardently long for an improvement. Nor am I singular in experience. Our surgical registrars tell us that in the decade 1892—1901 sixty cases of acute intestinal obstruction were admitted into the surgical wards of St. Bartholomew's Hospital, and that only fifteen survived.†

Intestinal obstruction must always be one of the most dire dangers that can befall a human being. Obviously a stoppage in the alimentary tract must be as fatal—though not so quickly fatal—as a stoppage in the air-passages or circulatory apparatus. But nevertheless, when I look back upon my own fatalities, I cannot but think that a proportion of them ought never to have occurred. Patients have been seen and operated upon after they had reached a desperate state. The delay seems mainly to have been due to uncertainty as to what "intestinal obstruction" is, hope that remedies might ultimately prevail, and doubt whether surgery could succeed. A certain sympathy must be felt for this frame of mind. The literature on intestinal obstruction is, for ordinary purposes, too full of the morbid anatomy of cases elucidated in the deadhouse. Such details ought to be familiar to the operating surgeon, but they are apt to overwhelm the practitioner; and, after all, it is he who is first called in to say whether the patient has or has not got intestinal obstruction. Then as regards the surgical treatment. It must be confessed that hitherto the results have not been encouraging; but the fatalities after operations cannot be attributed to a single cause, such as the operation itself, but to a combination of circumstances. To mention one circumstance, I would instance the want of clear plans for the management of the case from its beginning.

From the clinical standpoint most cases of intestinal obstruction pass through three phases. In the first the question is whether the patient has or has not got obstruction; in the second whether the obstruction is amenable to medical treatment; and in the third whether surgical measures should be tried. The chances of successful surgical treatment are oftentimes lost because the first and second phases are so prolonged.

Now let us try to answer these three questions; but before proceeding let me refer for a moment to the title of this communication. The word *recognition* is used because in so many cases recognition is all that can be achieved. A diagnosis cannot be made except by a surgical operation or post-mortem examination. Now the recognition of intestinal obstruction is a simple matter. It is safe to assume that anyone who cannot pass flatus or fæces has intestinal obstruction, and that death will ensue if neither escapes.

At first this proposition may seem rather extreme. A person with constipation oftentimes passes no flatus or fæces for hours or days. But it is not that he *cannot*, but merely that he does not. And how are we to tell whether one who does not pass flatus or fæces has constipation or obstruction? Let us put aside all these cases in which an obvious cause of obstruction is apparent—such as hernia, an intussusception, a tumour in the pelvis, or a carcinoma of the rectum—and take one in which there is no obvious cause. I myself am accustomed to put the question of the presence or absence of obstruction to the test by administering enemas. Enemas skilfully administered by a medical man are safe, and often give the clearest

* An address delivered October 2nd, 1902, at the Opening Meeting of the North-East London Clinical Society.

† The class of case under this heading is not defined, but cases of intussusception are given separately.

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information. Let me give some instances which can be recalled. An infant æt. 3 years was sent up with the intimation that she had intussusception. When seen the clinical signs fitted well with this diagnosis; no flatus or fæces had been passed for forty-eight hours. The abdomen was slightly distended; vomiting was frequent and persistent; the child cried out with pain, and put her hands upon the abdomen, where an obscure swelling could be felt. An enema was ordered, and arrangements made to open the abdomen if it failed. A motion followed the enema, and it ultimately became clear that the child had tuberculous meningitis. Doubtless the swelling felt in the abdomen consisted of tuberculous glands. I can also recall the case of an elderly lady who had had for many hours inability to pass flatus or fæces. Her breast was the seat of an ulcerated scirrhus, and her lower extremities were paralysed. The abdomen was extraordinarily tense and distended. The obstruction had been preceded by constipation, and had come on gradually. As there was no clear evidence of a permanent mechanical cause for the obstruction, and as there was a possibility of its being paralytic, enemas were tried whilst preparations were being made to perform laparotomy. It was evident that such severe obstruction would only yield to strenuous efforts, and therefore an anæsthetic was administered whilst the enemas were being given. A fair passage of flatus ensued, and afterwards Drs. Coleman and Colby completely succeeded in relieving the obstruction. I have notes of obstruction complicating paralysis agitans in a man 76 years of age. It was relieved by the long tube and enemas. Indeed, these measures may be more efficacious than colotomy in this class of case. For instance, a woman with secondary growths in the liver and peritoneum after carcinoma of the breast was dying of intestinal obstruction. I performed inguinal colotomy, but this gave incomplete relief, and she died on the fourth day. Afterwards no trace of a mechanical cause was discovered.

I believe that enemas given skilfully may be considered a test of the presence or absence of intestinal obstruction; when they fail, surgery is the next resort. As regards the composition of the enema, it will be found that soap and water mixed with turpentine and castor oil is very efficacious. The ingredients are easy to obtain—glycerine may be added, or sulphate of magnesia. In a case of obstruction following the opening of a large acute appendicular abscess, and in which the obstruction was known to be due to peritonitis and not to mechanical causes, an enema of soap and water, castor oil, turpentine, and three drops of croton oil succeeded when everything else had failed. Croton oil is very effectual, but only justifiable when it is known that there is no mechanical obstruction.

The enema is best given with a funnel and tube. Both the amount of force and the quantity can be judged more accurately than with the usual syringe. In addition, there is less chance of introducing air, which, of course, would return and be likely to mislead.

The temptation is great in cases of supposed intestinal obstruction to put the question to the test by giving purgatives by the mouth. I have learnt to look upon this as a most hazardous and dangerous proceeding, and a grave complication of surgical measures. By increasing the peristalsis of the intestines, purgatives excite pain and turmoil, and may increase the vomiting and exhaustion; the violent peristalsis which they excite is more likely to aggravate the effects of a mechanical obstruction than to overcome it; next, they fill the intestines above the obstruction with fluid and gaseous contents, and aggravate the abdominal distension; and last, they are a source of peril when the obstruction is relieved by operation. The following is not an uncommon history:—A carcinoma of the rectum is causing increasing difficulty in emptying the bowels. At first this yields to aperients, but at last a time comes when aperients fail and no flatus or fæces escape. Stronger aperients are given, and the abdominal distension is increased. Inguinal colotomy is performed. The sigmoid flexure is tight almost to bursting, all its coats inflamed, the peritoneal beginning to tear, the muscular paralysed, and the mucous ulcerated. An artificial opening has to be made forthwith. Enormous quantities of liquid fæces escape, and continue to flow until the patient succumbs. Inasmuch as the fæcal discharge does not take place by the natural passage this is not called a death from exhaustion brought about by purging, but is not infrequently attributed to the operation. Now the mortality from inguinal colotomy performed under favourable conditions

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cannot exceed 1 or 2 per cent., whilst under the foregoing it is extremely fatal.

On the other hand, enemas are safe. If they fail the fluid is on the right side of the obstruction, and merely comes back again. The faintness, vomiting, or the erythema which occasionally follow enemas need hardly be taken into consideration; neither a medical man nor an experienced nurse is likely to injure the rectum.

The test may be considered satisfactory if two enemas have been tried without anæsthesia and one during anæsthesia.

In intestinal obstruction the non-passage of flatus or of fæces is speedily followed by abdominal distension—with the advent of distension the gravity of the case is immeasurably increased. Its presence diminishes the chances of relief by enemas. As the late Grieg Smith argued, the tightly distended coils of intestine obstruct one another. Next, it is difficult or impossible after distension has supervened to localise the seat of obstruction. Even an intussusception becomes obscured, or an intra-peritoneal abscess, or a carcinoma of the cæcum or of the sigmoid flexure. Now all surgeons would agree that the chances of a successful operation are vastly greater when the abdomen is slack and when the operation has a clear objective, such as an easily felt tumour. Whilst performing laparotomy I have failed on more than one occasion to find the seat of obstruction, owing to the extreme intestinal distension. But now I feel less dread of extruding and emptying the distended bowels. The distension often necessitates that serious addition to the operation, namely, the incision and emptying of the distended coils. I have had to do this twice this year. An infant recovered after the jejunum and ileum had been emptied. An adult died after a similar proceeding, but his acute obstruction had lasted for eighty hours before it was relieved. Distension has also another evil effect. We are all familiar with the paralysis of the urinary bladder which follows retention of urine. After the stretching the muscular coats refuse to contract, and catheters have to be passed. Now the muscular walls of the intestine suffer in the same way from gaseous and fæcal distension, with the result that after the cause of the obstruction has been relieved by operation an adequate passage of flatus and fæces does not take place, vomiting continues, and the patient dies. When it seems probable that distension paralysis is established, a temporary opening has to be made in the cæcum or small intestine. Some time ago I operated upon a man with intestinal obstruction and enormous abdominal distension. He was too ill to allow of more than relief by an opening into the cæcum. A few days after this had been made his bowels acted and he recovered. I never heard the ultimate end of his case. Another man's life was clearly saved by an enormous escape of flatus and fæces from an accidental wound in the cæcum. This had been sutured, but gave way, owing to the extreme tension. Not long ago I had occasion to regret that I did not open and drain the small intestine. I saw with my friend Dr. Courtenay Drake a patient who had got an appendicular abscess and very severe abdominal distension. The appendicular abscess was with difficulty found and emptied. The appendix was then looked for by separating the ileo-cæcal junction. Whilst this was being done a quantity of gas escaped, and considerably relieved the distension. After the operation relief was obtained by enemas, but not in an adequate degree. I feel sure now that the opening at the ileo-cæcal junction ought to have been enlarged and a drain inserted. Owing to the fluidity of the fæcal flow, artificial openings into the cæcum and ileum are especially repulsive, and only to be made with extreme reluctance. Nevertheless they have a remarkable tendency to close themselves, provided there is a clear passage throughout the intestines.

I fear there are some who do not realise that when the passage through the bowels is obstructed flatulent distension is sure to occur. But a glance at colon bacilli growing in a tube of gelatine would speedily clear up any lingering doubt. At each colony of bacilli is a little bubble of gas produced by the bacilli themselves. The gas is evolved with speed, and continuously. Now think of the countless numbers of the intestinal bacteria and try to imagine the effects of their activity. No wonder the intestines distend so quickly, and no wonder the distension is so continuous and so uncontrollable.

Clearly the speedy onset of distension has to be taken into consideration in the management of cases of intestinal obstruction. If the patient be seen before it has supervened, the seat and cause of the obstruction may be ascertained. A tumour,

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for instance, may be felt in the sigmoid flexure, the colon, or in the right iliac fossa. And perhaps at this point I may be allowed to interject that the search for the cause of obstruction is sometimes pursued with a lack of method. I myself usually follow the course of the intestinal tract from the anus to the mouth. The rectal and vaginal examinations have demonstrated the presence of impacted fæces, of carcinomatous stricture of the rectum, of intussusception, of carcinoma of the uterus filling the pelvis, of fibroids of the uterus blocking the pelvis, or of extensive pelvic inflammation compressing the bowel. Also of collections of pus in the recto-vesical pouch or in Douglas's pouch. More than once I have felt the recto-vesical pouch filled with collapsed intestines, owing to mechanical obstruction near the jejunum. These collapsed intestines have a very peculiar feel, but their recognition at once shows that it is futile to attempt to give relief by opening any part of the large intestine; the obstruction is in the small. Median laparotomy is the only resource. After the pelvis has been examined, the course of the sigmoid flexure, left, transverse, and right colon are searched until the cæcum and ileo-cæcal junction are reached; then the area of the small intestines is felt, the region of the duodenum and stomach, and afterwards the liver and gall-bladder, and other abdominal organs. I am now more reluctant than some to dogmatise about what has been felt but not seen. In cases of obstruction upon which I have operated the digital diagnosis has often erred. A tumour, diagnosed by others as an intussusception, was a mass of acutely inflamed and suppurating mesenteric glands, undoubtedly tuberculous; and that which was supposed to be an acute appendicular abscess proved a suppurating ovarian cyst. But although the guess as to the nature of these tumours was wrong, the knowledge of their presence was invaluable, because it afforded a clear objective at which to aim.

I have as yet said nothing about vomiting as a complication of intestinal obstruction. Early, severe, continuous vomiting is seen in cases which run a rapidly fatal course. Its presence prohibits procrastination. I have seen the worst vomiting in cases of acutely strangulated umbilical hernia, the obstruction being high up in the jejunum, and very acute. Indeed, in such a case I had in 1898 to resect a gangrenous loop of bowel. The strangulation had probably lasted seventy hours. In spite of the unfavourable circumstances the patient recovered. Vomiting, too, was acute and persistent in the case of a young man who had a loop of ileum strangulated to gangrene beneath an adherent vermiform appendix. About forty hours of strangulation sufficed for the death of the gut, which had to be resected. A fatal result ensued on the fourth day. The cause was not ascertained. Likewise in volvulus of the small intestine—a rare event—I have seen early and continuous vomiting. In all of these instances the mere obstruction is not the only cause of the vomiting; the injury to the intestinal walls and mesentery and their nerve-supply is a factor, and helps to explain its sudden and violent onset.

Vomiting is a marked feature of a class of intestinal obstruction which in my experience is very common, namely, that which is caused by acute suppurative or non-suppurative peritonitis. Here, again, it is an indication of the dangerous severity of that which is causing the obstruction.

As an indication of peril the character of the vomit is immaterial. Sometimes in a desperate case those in attendance begin by remarking that fæcal vomiting has not set in, as though that in itself were a hopeful sign. All may yet be well if the vomit be not fæcal. Now, in itself the act of vomiting is exhausting and conducive to thirst. First food is ejected from the stomach, then mucus and gastric juice, then bile from the duodenum, grumous material from the jejunum, and at last yellow stercoraceous fluid from the ileum. One would have thought that affairs were becoming desperate long before the jejunal contents had been reached. Vomiting is a distinct addition to the dangers of anæsthesia. I believe some patients die because during unconsciousness vomit enters the air-passages. But I have become afraid of trying to obviate this danger by washing out the stomach. The passage of the tube and subsequent washing has, in the cases in which I have tried it, caused too much exhaustion and collapse. Now this is a very serious matter when a prolonged and dangerous operation may have to be performed. On the whole, the danger of vomiting is less to be feared when the anæsthetist is skilful and does not push the anæsthetic too far, and takes care to keep the head low and well on one side.

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I have purposely said nothing about pain or tenderness. Neither of them is indicative of obstruction, but either may afford some slight clue to the seat of obstruction. Pain, however, is most deceptive. Tenderness accompanied by muscular rigidity is more to be depended upon. But pain has one very evil result. It often leads to the administration of opium or morphia. When a human being is in agony it may seem hard to withhold relief, but the narcotic is sure to accelerate the onset of the intestinal paralysis and gaseous distension. The pain ought to be relieved by the division of the band, the withdrawal of the intussusception, or the evacuation of the abscess.

In the examination of a case of intestinal obstruction it is of course helpful to have in the memory a classification of its possible causes. An anatomical classification, such as is usually given, may be of use in the arrangement of museum specimens, but is not very serviceable at the bedside. My own experience has led me to classify the causes of intestinal obstruction into—(1) mechanical, (2) inflammatory, (3) paralytic, (4) vascular. The last is the only division of which nothing has yet been said. Once I performed laparotomy for acute intestinal obstruction, and found thrombosis of the mesenteric veins. On another occasion the mesenteric arteries seemed to be at fault, having possibly become filled with clot. But even such a simple classification as this has to be used with caution. Take such a case as the following, and one neither unusual nor imaginary. A young woman was suddenly seized with violent abdominal pain, ceased to pass wind or fæces, vomited, and began to get distended. No cause could be discovered. The intestines were soon in a state of violent peristalsis. With a stethoscope gurglings and "gugglings" could have been heard all over the belly. The temperature was normal. The pulse 85, and of full volume. The skin was moist. After a while the intestines became stiller, and the distension markedly increased. Then at last the abdomen was very still and quiet. Nothing was heard with the stethoscope. The skin was now dry, the temperature slightly raised, but the pulse 140 and difficult to count. What is the interpretation of such a case? First, a mechanical obstruction; second, a huge peristaltic effort to overcome the obstruction—with the pulse and perspiration which accompany exertion; third, muscular exhaustion and paralysis with dilation of the intestines; fourth, peritonitis starting at the strangulated loop, which had by that time become gangrenous. As I have already pointed out, the presence of peritonitis causes the most absolute intestinal immobility. I always consider it a hopeful sign if vermicular movements can be seen before the operation is begun. It is clear evidence of the absence of paralysis and of peritonitis.

We now come to the second part of my communication, namely, the management of a case of intestinal obstruction. Some of the most essential points have already been touched upon. And first it is clear that, inasmuch as intestinal obstruction is inevitably fatal, there should be no pause or cessation in the endeavour to procure relief. I am of opinion that it is the duty of medical men to remain with the patient until relief is obtained. I am fully aware that some may be startled by this proposition, but see how it works out. The patient has passed no flatus and no fæces for some hours or days. Perhaps some of the other signs of obstruction are added. An enema is given, and fails; a second likewise fails. Aid is sent for at once, and an anæsthetic is given; possibly another enema is tried, and fails. Then laparotomy is performed under the most favourable conditions, that is to say, before intestinal distension and paralysis, and, of course, long before the peritoneum is inflamed and cracked, or the mucous membrane ulcerated, or before gangrene has supervened. When this line of management cannot be carried out at the patient's home, an immediate start should be made for the hospital or nursing home.

In about half the cases, by systematic investigation, cause can be found to explain the obstruction. This cause has forthwith to be dealt with. For instance, fæcal impaction may be found, and relieved with enemas. If these are unsatisfactory, they may be tried again after an anæsthetic has been given. We have already had instances of the success in cases of paralytic distension. But obviously obstruction caused by growths in the bowel, or by pelvic inflammation, is not likely to yield to measures such as these. Surgery ought to step in without delay. When the administration of an anæsthetic has been decided upon, then proper preparations should be made for the performance of an operation. As I have already said, the gravity of the operation depends in a large degree

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upon the stage at which it is performed. In the absence of distension the abdomen can be systematically explored and the seat of the obstruction accurately ascertained. Accurate knowledge upon this point is of essential importance. Should the distension be so great as to prevent a proper examination, then I believe it is justifiable to extrude the distended intestines through the median incision, and empty them of their fæcal contents. After this has been done a proper search becomes possible. This ought to be conducted in a routine manner; first the ileo-cæcal junction, then the colon, sigmoid flexure, and lastly the small intestines. Sometimes collapsed intestine at once comes into view, and is a guide to the seat of obstruction. I have been entirely deceived by trusting to mere distension of the intestine as a guide to the seat of obstruction. Once it was assumed that the obstruction was high in the rectum; the sigmoid flexure was exposed, found distended with gas, and fixed in the abdominal wound and opened. Gas and a little fæcal matter escaped, but it was afterwards ascertained that the obstruction was due to volvulus of the small intestine. On another occasion it was assumed that the obstruction was in the hepatic flexure of the colon; the distended cæcum was opened. At a subsequent operation, when the abdomen was no longer distended, the carcinomatous stricture was found high in the rectum. The cæcal opening was a source of discomfort, and the large intestine got loaded with fæces, so that sigmoid colotomy had to be performed. Whilst one can look back upon a few cases in which a happy guess has been made as to the seat of obstruction, one has also to confess that there are many others in which the guess has been wrong. In the absence of some guiding sign, it seems hardly safe to infer more than that in chronic intestinal obstruction the cause is in some part of the large intestine, whilst in acute it may be in the small. Hitherto I have purposely avoided the use of the terms *acute* and *chronic*. The assumption that a case is chronic oftentimes tends to fatal delay. Chronic cases may at any moment become acute, and even in the chronic irreparable changes are slowly but surely progressing. When the cause of the obstruction has been located it may be easy to decide what to do; but some of the problems which arise are full of embarrassment. I have already mentioned a case in which, after the removal of the cause of the obstruction, an error of judgment was probably committed in not immediately draining the small intestine through a temporary opening. After the intestine has been distended it seems as though it cannot be relied upon to propel its contents through the part of it which has been occluded. I remember a desperate case which was under the care of Dr. Wunderlich and Dr. Hutt, and in which acute intestinal obstruction was caused by injury to some coils of small intestine matted together by a long past attack of appendicitis. In this case median laparotomy was performed, and the distended intestines extruded and emptied, and afterwards the matted intestines separated until they became filled with gas. I assumed wrongly that the passage of gas would continue and give relief. Thinking the matter over, three other courses were open: first, a temporary opening might have been made above the entangled mass; second, the intestine above might have been anastomosed with the colon; and third, the matted intestine might have been excised. On the whole, the second of these alternatives seems to offer the best chances of success, and I regret that I did not adopt it. The question, too, of the excision of carcinomatous strictures has to be decided. As a rule, owing to distensions and paralysis, it is advisable to give relief by opening the bowel, and then seize a favourable opportunity for excision. Other problems arise when intestinal obstruction is caused by tumours such as those of the uterus. I remember assisting my colleague, Mr. Harrison Cripps, to remove a fibroid tumour of the uterus under these circumstances. Here, again, the intestines which had been distended were unequal to the propulsion of their contents after the fibroid tumour had been removed. The patient's life was saved by the prompt performance of typhlotomy. Few surgeons could hope to be so successful under such desperate circumstances. In some classes of case it seems now to be well recognised throughout the profession that prompt measures afford excellent chances of recovery. This is notably so with regard to strangulated hernia and intussusception, but it is clear from the figures which I have given that intestinal obstruction in which the obvious cause is not discovered does not yet receive the same prompt attention.

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FÆCAL FISTULA COMPLICATING TUBERCULOUS PERITONITIS; TRANSVERSE COLOTOMY; ACUTE INTESTINAL OBSTRUCTION; DEATH.

By LAWRIE McGAVIN, F.R.C.S.,
Assistant Surgeon to the Hospital for Women, Soho Square, W.

On July 31st, 1901, a young girl æt. 18 was transferred by my colleague to my care, suffering from a fæcal fistula, of which the following is briefly the history.

In the previous April she had been operated upon for a double pyosalpinx, which condition appears to have originated about twelve months previously, she having had an attack of abdominal pain at that time, since when no menstrual periods had occurred. At the operation the pelvis was found to be occupied by a large right pyosalpinx about the size of an ostrich's egg, the left tube being also affected, but to a less extent, and the appendages matted and adherent to the intestines. The pus was extremely offensive, but no communication with the bowel was found. The ovaries and tubes were removed, the left ovary containing some cysts, in which caseous material was found; similar caseating masses were present in the broad ligament. On removal of the stitches on the eighth day a quantity of feculent pus welled up in the centre of the wound, and from that time onward almost the whole of the patient's fæces passed in this way. On the date of transfer this patient was in an extremely emaciated condition, and had a decidedly hectic appearance; the abdomen was markedly scaphoid, the limbs wasted, and the iliac spines prominent; in addition there was evidence of the early appearance of bedsores over the sacrum. Occupying the situation of the old wound, midway between the pubes and the umbilicus, was an area of granulation tissue measuring three inches by one, and at the lower third of this was the opening of the fistula, large enough to admit the tip of the finger, and into which a probe could be passed in the direction of the left sacro-iliac joint for a distance of four inches. This could not be felt *per rectum*, although its movements could be detected through the intervening tissues. The mouth of the fistula was extremely tender to the touch, and excretion was carried on almost entirely by means of the fistula, very little fæcal matter coming from the anus.

The appearance of the excreta, which were semi-solid and of a healthy colour, suggested the large intestine as the probable site of the enteric opening, and the direction of the sinus and the previous history of the case supported this view. At the urgent representation of the parents, who were duly warned of the serious nature of the proposed operation, I performed laparotomy on August 2nd, hoping to be able to short-circuit the bowel, or possibly to excise the track.

On examining the organs a most extensive condition of tuberculous peritonitis was found, the whole of the small intestine being studded with miliary tubercles, many of which were of the size of grains of rice. The upper half of the abdomen was comparatively free from adhesions, while the pelvis and lower half of the cavity were the seat of dense adhesions, the attempt to separate which gave rise to free bleeding. The sigmoid, the cæcum, and the lower few inches of the ileum were involved in these adhesions, and the uterus could not be made out.

Under the circumstances, the attempt to excise the track or to short-circuit the bowel was necessarily abandoned, and I determined to perform a transverse colotomy which would, I hoped, exclude the fistulous track, and in the event of its subsequent closure enable me to reconstruct the bowel more easily in this situation than in the lower part of the abdomen. The colotomy was carried out by Maydl's method, the transverse colon being drawn well to the left to avoid the chance of prolapse as much as possible. The whole abdomen was then washed out with sterilised water, of which a considerable quantity was left in the cavity. The colon was opened on the fifth day, the rod being removed on the seventh. Much relief was the result of the operation, the sinus becoming smaller, the tenderness almost disappearing, and the patient gaining slightly in weight.

The colotomy wound worked admirably, and all went well till three weeks from the date of the operation, August 22nd, when fæces again appeared at the mouth of the sinus in considerable quantity. It was possible at this time to wash the distal portion of the bowel out by syringing from the colotomy wound, the fluid all escaping at the sinus;

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but the like result could not be obtained in the reverse direction, although the reappearance of fæces at the sinus pointed to the existence of a second enteric orifice, probably either in the ileum or cæcum.

A light spring truss was now fitted, with some difficulty, to the mouth of the sinus, and pressure applied in this way, the track being washed out each morning and evening. The fistula now showed every indication of closing, the mouth being reduced to about the diameter of a lead pencil, and but little fæcal matter coming through; the patient was accordingly permitted to go out of doors each day.

On October 31st, while walking in the gardens in front of the hospital, she was suddenly seized with severe abdominal pain, and on returning indoors commenced to vomit (half an hour after the first attack of pain). When seen about four hours later she was in great pain, with marked abdominal aspect, and considerable tenderness over the upper part of the abdomen. In spite of the evident acute obstruction she was still passing fæces and flatus from time to time.

The parents of the patient, on being sent for, were unwilling that anything further should be done, and in view of her extremely emaciated and tuberculous condition, and the very doubtful possibility of finding the seat of the trouble in an abdomen now so completely disorganised, I did not strongly urge the matter. The whole course of the strangulation was very acute, for the vomit became fæcal on the second day, and the patient sank and died on the fourth. The following is the report of the autopsy, which was performed by Dr. Norman Dalton.

The emaciation was very marked. On the mitral valve were a few small warty vegetations; in other respects the thoracic organs were normal. Liver, spleen, and kidneys normal.

The scar of the previous operation lay in the middle line of the abdomen. At its upper end there was an opening which was subsequently found to communicate with the transverse colon. About the middle of the scar there was an ulcer, about the size of a glove button, and partly scabbed over. This ulcer did not extend into the peritoneal cavity.

In the lower part of the scar there was an opening which led both into the cæcum and into the sigmoid. The parietal peritoneum was sparsely sprinkled with tubercles, but not to the extent noted at the operation, and none were found on the intestines. There was no general adhesion of the peritoneal surfaces, so that the comparative cure of the visceral tuberculosis cannot be attributed to the formation of adhesions.

The opening into the transverse colon was surrounded by old fibrous tissue, and that part of the bowel was collapsed.

The lowest opening in the abdominal scar led into the sigmoid, but it was found that a part of the cæcum beyond the ileo-cæcal valve opened into the sigmoid at the same spot, and consequently communicated with the abdominal fistula. This fistula and the adjacent pieces of bowel were quite cut off from the peritoneal cavity by old fibrous adhesions, and the whole of the pelvic peritoneal cavity was obliterated by dense adhesions in which it was not possible to find the uterus.

The stomach and the first four feet of the small intestine were greatly distended, the remainder being collapsed. Some of the distended coils crossed the abdominal cavity between the adhesions at the back of the wound. On tracing the distended part of the bowel downwards from the duodenum, the point of obstruction was found, and proved to be a fibrous band, rounded, and about the size of a quill pen. At one end it was attached to the peritoneum over the left psoas muscle, and at the other to the anterior abdominal wall near the laparotomy scar, passing round a coil of the jejunum. It was about three inches long, and the constriction, though sufficient to block the lumen of the gut, was not very tight, nor did the constricted portion of the bowel show any marked degree of peritonitis. The site of the strangulation lay much deeper in the abdomen than this account would seem to indicate, and it was considered that the chance of relieving the condition during life would have been small, for the constriction was only found, and the coils of bowel around it unravelled with some difficulty, even at the autopsy.

My reason for putting this case on record is threefold. Firstly, acute intestinal obstruction following on tuberculous peritonitis is not so commonly seen as the condition of the abdominal cavity in this disease would seem to render probable. And this is possibly accounted for by

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the fact that peritoneal tubercle, being more commonly diffuse than localised, is more prone to produce a general binding down of the intestines, and therefore a limitation of their peristalsis, than one or two isolated adhesions, which, becoming stretched, either ensnare a loop of bowel, or allow such a loop to pass under or over them, and thus to become strangulated. The question might be raised as to whether the adhesion found at the autopsy arose from the peritonitis, or was the direct result of the operation. As against this latter I would place the following points:—The portion of the wound which was nearest the parietal end of the adhesion healed by primary union; the visceral end of the adhesion lay at some considerable depth from it, viz. on the sheath of the psoas muscle; it is unlikely, therefore, that those two parts were ever in close enough apposition to permit of the formation of an adhesion which subsequently became stretched; the adhesion involved that part of the intestine which was found at the operation to be most actively tuberculous; and, lastly, the parietal end was not attached actually to the scar, but rather to one side of it.

Secondly, the extraordinary disappearance of the tubercles, previously so numerous, from the intestines, although at the present time a well-known result of laparotomy in such cases, was yet in this particular instance so remarkable and so rapid, that had the case been only one of peritoneal tubercle, and uncomplicated by fistula, it would have been worthy of being placed in the same category with the best results which have so far been reported in this connection. For, as far as the intestine was concerned, not only had the largest of the tubercles disappeared, but even their sites were no longer indicated by cicatrisation or anæmic patches. Only on the parietal peritoneum were there any tubercles found, and of these none were larger than a pin's point, and they, moreover, were only present at one spot. So striking was the change, that under other circumstances I have little doubt but that, as far as the tuberculous peritonitis is concerned, a complete resolution of the disease would have resulted.

Thirdly, the operation of transverse colotomy is, as pointed out by Mr. Jacobson,* the least frequently performed of all the colotomies; and the reason for this is obvious, for where the sigmoid is healthy it is undesirable to cut off any greater length of the alimentary canal than that which lies between the sigmoid and the anus. But where the sigmoid is diseased, and is adherent, malignant, or, as in this case, fistulous, the transverse colon will be found to answer the purposes of colotomy very efficiently. For although Mr. Allingham* draws attention to the fact that the motions are more difficult to manage, owing to their greater looseness, in the transverse than in the descending colon, the method has, I think, the following advantages, viz. prolapse is less probable, owing to the more horizontal position of this portion of the bowel, and to the firmer support afforded by the abdominal wall in the epigastric and umbilical than in the inguinal region. An excellent spur is obtainable, owing to the convenient and fairly constant length of the transverse mesocolon, a result not always to be obtained in the sigmoid where the mesosigmoid is often unduly short; and this is a most important point in dealing with very stout patients. A good belt and pad is rather more easily fitted here than in the case of the sigmoid, and certainly has a more resistent part on which to act than when placed on the resilient and flexile inguinal region; leakage around the plug is therefore less troublesome than in inguinal colotomy. Finally, should reconstitution of the bowel be desirable at a later date, it is certainly more easily carried out here than in the sigmoid.

* 'The Operations of Surgery,' chap. iii, p. 112.

* 'Diseases of the Rectum,' chap. xxxi, p. 443.

Toothpick Method of Treating Lupus.—Richter has slightly modified Unna's original method of treating isolated lupic nodules. He uses toothpicks made of beechwood impregnated with the Liq. Stibii Chlorati, which takes up the fluid well and penetrates readily into the lupic lesions, but is arrested by sound tissue. The necrosed plug drops out in three or four days, and he then fits a plug of 33 per cent. orthoform cotton in its place. The cotton after fifteen minutes is then moistened with one or two drops of the same caustic, and the spot covered with plaster. In two to four days more the desired necrotisation is complete, and the patch heals under sublimate-lead water. For nodules on the nose or near the eyes, a thorn answers better than a toothpick, as it is less painful.—*Journ. A.M.A.*, September 27th, 1902.

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* *Specially reported for The Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE

ON

WHOOPING-COUGH.

Delivered at the Hospital for Sick Children, Great Ormond Street, W.C.; June 5th, 1902.

By A. F. VOELCKER, M.D., F.R.C.P.,

Physician to the Hospital, and Assistant Physician and Lecturer on Pathology, Middlesex Hospital.

LADIES AND GENTLEMEN,—I am sorry I cannot invite you to come up into our ward, for the very good or very bad reason that we have had an outbreak of diphtheria and a case of scarlet fever in the ward, which therefore has been closed, and such cases as could go out have been sent out, so that practically there is nothing to show you in the ward. At the close of my remarks I propose to demonstrate to you, in the post-mortem room, the viscera of one of the children who has been under my care, and who has died with extensive tubercular lesions.

At the present time, when the public conscience is being roused to a better sense of its undoubted duty and responsibility in the matter of contributing to the support of institutions and of individuals engaged in the investigation and treatment of disease and of matters affecting the health and welfare of the community at large, it is satisfactory to find that in addition to the generous contributions made to the general funds of this hospital, the Goldsmiths' Company have, by their handsome liberality, enabled us at this hospital to open a ward of sixteen beds into which cases of whooping-cough are admitted for treatment. This ward has now been open twenty months, and it has been my good fortune to have had charge of the ward all the time. The position of unusual opportunities or facilities for the observation of any particular disease or class of diseases lays the possessor under the moral obliga-

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tion of endeavouring to add to the sum total of our general knowledge on the subject, even if the acquired information be only of the nature of confirmation of the opinion of others. In bringing before you some of the results of my observations on the cases under my care I must claim your indulgence for a repetition of many facts well known to many of you; and if after all I am not in a position to answer the question which seems to rise spontaneously to the lips of so many people, "What is the cure for whooping-cough?" yet I hope to be able to point out to you some of the factors concerned in the successful treatment of the disease, some of the complications and their import, and some points which have proved of practical utility in the diagnosis of this disease. I may say at once I do not intend to give you a discourse upon whooping-cough; that is a thing which you can get out of books, and many of you probably have had considerable experience of it, and many have had opportunities of reading about it. What I wish to do is to put before you my own personal experience of the cases treated in my own ward. At the outset I should like to insist on an important point, and that is that the remarks I am about to make refer to the special cases under my care, and not to cases of whooping-cough as a whole. Owing to the infective nature of the disease, and to the great amount of attention which severe cases of whooping-cough require from the nursing staff, and from the fact that there are only two hospitals in London into which cases of whooping-cough are admitted as in-patients, the cases which come under our observation are naturally very severe ones, and in the greater number of instances are, at the time of their admission, suffering from some complication, most commonly broncho-pneumonia. The claims of these severe cases make it almost impossible for us to admit uncomplicated cases of whooping-cough; but, on the other hand, we get from time to time cases of relatively mild whooping-cough which come in because they are suffering from some other disease, most commonly some surgical complication which requires treatment in a hospital, while the infective nature of the disease renders it impossible to carry this out in the general wards of a children's hospital. So you will remember that the majority of our cases are extremely severe cases, generally complicated, while a certain number are relatively mild cases, in which the whooping-cough is simply, as it were, an accident in the course of some other disease. This has an important bearing on the frequency with which we find complications, and also on the effect of treatment.

It is not necessary, I am sure, to define whooping-cough to you. The first point I wish to speak of is its infectious nature. That it is infectious is well known, but from the fact that we only take in the severe cases it is rarely that we have as in-patients more than two children of one family at the same time. I think since the ward has been opened I have only had on three occasions children from the same family as in-patients at the same time. The infectivity of whooping-cough cannot be very great, because we find that although cases are sent to us from the general wards of the hospital, cases which have been in for some time and have then developed undoubted whooping-cough, yet we have not had more than one case ever sent from any one ward. Now if the disease had spread in the way in which we find measles or diphtheria spreading, we should have had more cases sent in from one ward. Again, the only instances in which I have had runs of cases have been from Convalescent Homes, or in bedridden children. Another point with regard to the infective nature of whooping-cough is that while our nursing staff in the ward is separate—that is to say, that the nurses do not nurse in any other ward, yet the sisters and nurses of this ward have their meals in common with the other sisters and nurses of the general hospital. In spite of this we have had no case of whooping-cough arising in the hospital which we could trace to infection conveyed by any one in the hospital itself. On this question of isolation of nurses we take an entirely different course from the one we adopt with regard to our treatment of other infectious cases, for which we have an entirely separate staff, which has nothing to do with the rest of the nursing staff of the general hospital. Those points show us that although whooping-cough is infective, yet with suitable hygienic precautions, that is to say plenty of air space and good ventilation and ordinary cleanliness, it is possible for us to successfully isolate cases of whooping-cough.

The *diagnosis* of whooping-cough does not often come before us as a very practical difficulty.

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Still, there are some points which deserve attention in cases of suspected whooping-cough. The cases that are most likely to be mistaken for whooping-cough are cases either of enlarged bronchial glands pressing on the trachea or main bronchi, cases of bronchiectasis, cases of pleural effusions, cases of enlarged tonsils and adenoids, and occasionally a case of laryngismus stridulus. With regard to the differential diagnosis, careful physical examination is the essential thing in distinguishing these conditions, but I do not propose to go into the differential diagnosis of whooping-cough now. There are, however, certain points in connection with certain symptoms of whooping-cough which are perhaps worthy of your attention. First of all there is the *cough*. If the cough is paroxysmal and unattended by abnormal physical signs in the chest and pharynx, the probability is that the case is one of whooping-cough. If a cough is brought out by psychical disturbance, or as a result of mechanical stimulation of the pharynx, and then that cough is paroxysmal, the case is probably one of whooping-cough. The practical point is that when in doubt about a case of whooping-cough never neglect an examination of the throat. The more you have to do with children the more you will look at their throats. When in doubt as to the nature of a cough, or if you have reason to suspect the presence of whooping-cough, examine the child's throat and use a spatula. Do not be in too great a hurry, and finish up with a little stimulation of the back of the pharynx with the spatula; this will very often bring out a characteristic paroxysmal cough, or even induce a whoop which had hitherto been latent or unnoticed. It is said that the cough of whooping-cough is more frequent at night. Our experience in the ward is that it is not. The cough of whooping-cough, as seen in private, undoubtedly does show itself most commonly towards evening, and the reason is that in all probability it is then that the child is most tired, and, feeling ill, resents being undressed, cleaned up, and put to bed. It is probably partly the mental and partly the physical upset that make the cough more frequent. In the wards the paroxysms of coughing and the paroxysms of whooping-cough are distinctly more frequent in the daytime than they are at night. I think the explanation here is that during the daytime the children, if they are well enough, are playing about; they are also being somewhat disturbed by being cleaned and fed and having their temperatures taken; and I think pretty largely on this account, and partly because they have very good nights, we get less frequent paroxysms of coughing between 8 p.m. and 8 a.m. than between 8 a.m. and 8 p.m., those being the hours between which the number of whoops and coughs are charted, and the chart on which they are registered I will show you afterwards. Another point that has come out with regard to the frequency of whoops and coughing is that while the number of individual whoops varies tremendously—in the marked cases we have had 300 to 400 whoops in the twenty-four hours,—yet even in these very severe cases the number of paroxysms of coughing rarely exceeds twenty in the twenty-four hours. In no single case have as many as thirty paroxysms of coughing been noted in the twelve hours. Now it must be remembered, of course, that the frequency of the cough is no measure of the severity of the attacks of coughing. At the present time we have a child in the ward who has not very frequent attacks of coughing—I counted, a few mornings ago, twenty-four whoops to a single paroxysm of coughing; and although this would only count as one in the record of coughs while it would count as twenty-four in the record of whoops—yet those attacks were so severe that the child actually went into a condition of opisthotonos in the attacks of coughing. The child was perfectly blue about the face and extremities. Next to the coughing and the whooping is the question of *expectoration*. It is an aphorism that children do not spit,—that is to say, young children never spit. This aphorism, like everything else in medicine, displays the foolishness of employing the words "never" and "always" with regard to any phenomenon of disease. We might, for a moment, just consider conditions under which children do expectorate. The most common conditions which cause children under twelve years of age to expectorate are whooping-cough and bronchiectasis. In bronchiectasis the expectoration is most abundant in the early morning, on the patient first getting up; and it is frequently, but by no means universally, offensive. You must not, therefore, wait for the occurrence of offensive expectoration in a child before you diagnose a condition of bronchiectasis, if the physical signs justify that diagnosis. In diagnosing bronchi-

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ectasis, besides the physical signs in the chest, a very important point is the question of clubbing of the fingers, and a large number of cases of bronchiectasis occur in children who are the subjects of syphilis. Spitting, in the case of a child who has a cough, is very suggestive of whooping-cough. The mucus is very thick and tenacious, and it clings about the mouth in a striking manner. Hæmoptysis, which is not a common phenomenon in children, is met with fairly frequently in whooping-cough, although in the cases under our observation in the hospital it has been by no means common. Hæmoptysis in pulmonary tuberculosis in children is, as you know, rare. We have had in the whooping-cough ward one fatal case of hæmoptysis in a child with whooping-cough, in whom there was pulmonary tuberculosis, the lungs having been extensively destroyed by the disease.

Vomiting.—This is a frequent symptom of whooping-cough, and the act of vomiting is usually initiated by a cough. A combination of coughing and vomiting is very suggestive of whooping-cough, though by no means diagnostic of it. In bronchiectasis, in cases of enlarged tonsils, and in cases where we have enlarged bronchial glands, we may get co-existing coughing and vomiting. Vomiting occurred in over ninety per cent. of the cases of whooping-cough under our observation.

Sublingual ulcer.—This is a very suggestive sign of pertussis. The sublingual ulcer is an ulcer situated at the frænum, where the frænum joins the under surface of the tongue, and is usually single. It varies in size. It may be three quarters of an inch long by half an inch broad. It may be only the size of a lentil, or even smaller. But although usually single they are sometimes multiple. The ulcer is the result of mechanical injury. It is said only to be present in children who have cut their lower incisor teeth. This is not absolutely correct I have seen cases of sublingual ulcer in children who had no teeth at all. The explanation in these cases is simply that we have the under surface of the tongue and frænum markedly stretched over the alveolar margin; and although we have not a tooth there, we have sufficient stretching to cause laceration, and the constant movement prevents the healing. An interesting point about a sublingual ulcer is that it is very rarely painful; if we remember how painful aphthous stomatitis is in a child, it is curious to see a great big ulcer under the child's tongue, and yet the child practically free from pain. Perhaps in looking for the ulcer in children one is most forcibly reminded of the truth of the unruly nature of the tongue. It is an extremely difficult thing to get a child to put its tongue out so that you can look under it, and so to arrange things as to see what there is under the tongue. With regard to the frequency with which we meet sublingual ulcers, I have notes of 102 cases in which it was stated either to be present or absent. It has not been recorded in every case. But in the 102 cases in which it is mentioned it was present in thirty-four. That is roughly about one third of the cases. My house physician, Dr. Bull, has kindly noted for me its presence or absence in 136 cases which have come under his care in the casualty department, and he finds it is present in nearly 28 per cent. of the cases. The cases I have, of course, are the more severe ones. In the casualty department there are many cases of a mild character.

Age is an important factor in connection with the occurrence of the ulcer. It is extremely rare in infants under one year of age. In thirty-five cases in my ward in which it was present, only one child under one year of age presented it. This was in a child who had cut its lower central incisor teeth. It occurred in twelve cases between one and two years of age; in seven between two and three; in five between three and four; in six between four and five, and after this its occurrence was rare, but the majority of children in the ward are under six years of age. It must not be concluded that in the bigger children we did not get them; the point is that we did not see so many big children in the ward. Dr. Bull has found in the twenty-eight cases in which the ages were noted, nine occurred under two years of age, fourteen between two and five, and five above the age of five. From these observations it will be seen that the ulcers are most common in the second year of life as far as our observations have gone. Now is sublingual ulcer pathognomonic of pertussis? Probably no. I can call to mind only one case in which I have seen a sublingual ulcer and no pertussis, and that was in this hospital. Dr. Still tells me he has seen the condition present in a child who had severe paroxysmal coughing from enlarged bronchial glands, and only yesterday he tells me he saw a

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case of broncho-pneumonia in which there was a typical whooping-cough ulcer. Personally, then, I should be inclined to attach very great importance to the presence of a sublingual ulcer, though I cannot say that it never occurs in any other condition.

The next point is *subconjunctival hæmorrhage*. Subconjunctival hæmorrhage was not at all frequent. Out of 161 cases we have only had three cases in which subconjunctival hæmorrhages were noticed. Dr. Bull tells me that among 130 out-patients he noticed it in five cases. The hæmorrhages are not pathognomonic of pertussis, although suggestive. One point of interest is their position when they are extensive. They usually occupy the equator of the globe of the eyeball, but this equator is not a horizontal one. I think the reason of this is that the subconjunctival hæmorrhages occur in the parts that are the least supported, and the part which is least supported is naturally the part between the margins of the lids. From this we might expect that the hæmorrhages would be along the horizontal equator; but if we remember that in the act of coughing the eyes are turned upwards and outwards, I think we shall see we have an explanation of the fact that the equator will then come to lie obliquely, because the palpebral fissure remains horizontal. The eye moves, but the palpebral fissure remains horizontal. When the eye at rest is open—that is to say, the eyelids are opened,—this equator will be seen not to lie horizontally, but obliquely. I have one case which caused me to suggest that explanation, one marked case in which this obliquity was extremely definite and symmetrical. One point to remember about the hæmorrhages is that they are more extensive anteriorly than posteriorly, forming a contrast to the distribution of hæmorrhage which we get when we have lesions due to fracture of the base of the skull.

The last symptom is the *whoop*. The whoop gave the name to the disease. The disease was described by Willis in 1667, and the name which he gave it was "chin cough," and I believe that in some parts of England it is still known as the chin cough. With regard to the whoop, "one swallow does not make a summer," and one whoop will not make whooping-cough. We get cases of whooping-cough without any whoop, and we may get a whoop without any whooping-cough. In very young infants it is rare to get a whoop. But after six months of age whooping is not so infrequent, and we have had some very severe cases of whooping-cough in infants under one year of age. I have known enlarged tonsils, enlarged bronchial glands, bronchiectasis, pleural effusions—either purulent or serous,—accompanied by an occasional whoop; and I have known the crow of laryngismus stridulus mistaken for the whoop of pertussis. In the out-patient department some years ago I was astonished to see an interesting case of mine put aside. The mother of the patient came to me and said her child was said to have whooping-cough. I knew it was an old case of mine which had had marked rickets and tetany, so I waited and got the characteristic crowing, and then, of course, allowed that child to come in with the other out-patients. Only this spring—for spring is the time when the young child's fancy lightly turns to thoughts of *tetany*—we had no fewer than five cases of tetany in the ward, three of them with laryngismus stridulus and whooping-cough at the same time. That it is possible to tell the difference is evident from the fact that in going round the ward I pricked up my ears, metaphorically, on hearing a certain sound, and said "laryngismus stridulus"; and it was. Still the whoop has been mistaken for the crow of laryngismus stridulus. There are differences between the two sounds, and when one has heard a number of cases of each it is not difficult to detect them apart. When we come to examine a case and find, as we almost invariably do, the co-existence of facial irritability (a phenomenon in which stroking or tapping the skin over the region of the facial nerve is followed by contractions of the muscles of the mouth and eyelids) with twitching of the side of the face and that characteristic position of the hands in tetany—the accoucheur's hands, which we may elicit by compression of the vessels and nerves going to the limb (Trousseau's sign),—and if we find also marked rickets, there is very little difficulty in deciding that a given case is one of laryngismus stridulus, and not one of whooping-cough. So much for the symptoms.

The next point that has struck me is the influence of a rise of *temperature* on the frequency of the whoop. In a very large number of cases when the temperature rises the frequency of the whoop falls, and when the temperature falls the

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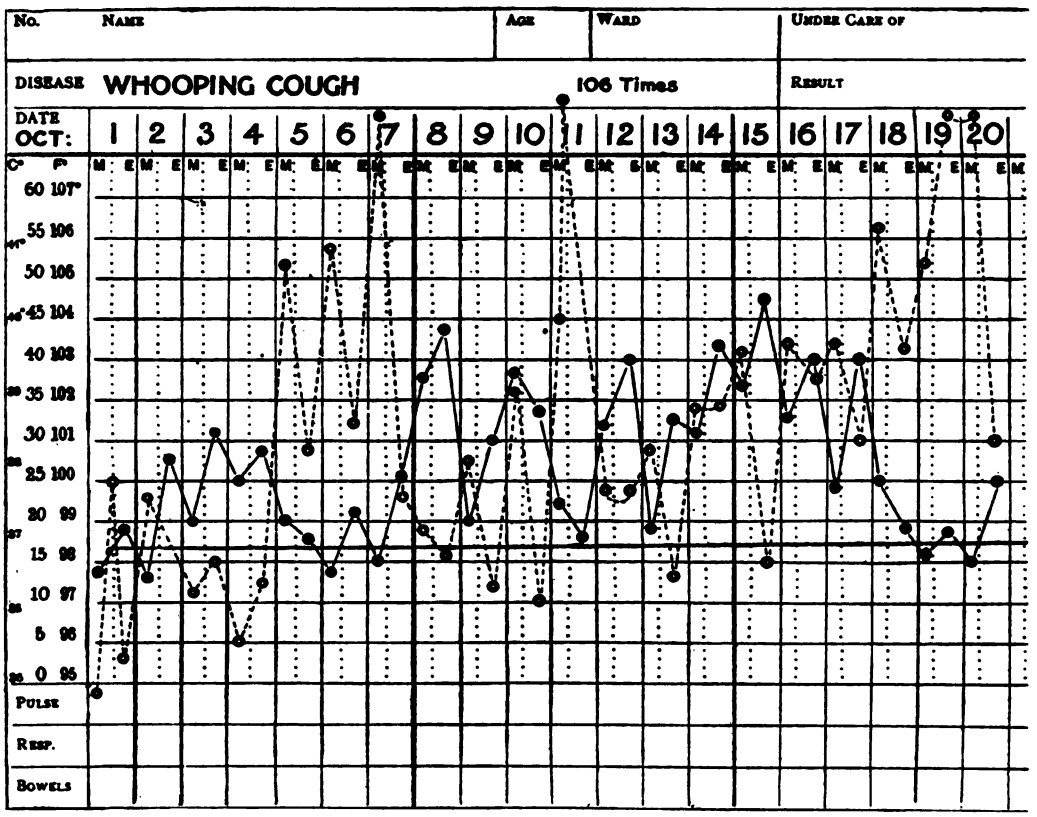
whooping increases in frequency. Here I show you part of a chart copied from a case in which this is very well shown (chart No. 1). This portion of the chart shows very well that when the temperature goes up the frequency of the whoops diminishes. And this is interesting because we find that, with the advent of acute inflammatory mischief, attacks of broncho-pneumonia or lobar pneumonia, or other inflammatory trouble, there is an apparent improvement sending in such a mild case. But I think these cases are very interesting, because I believe they indicate *collapse of the lung*, and in my experience the diagnosis of collapse of lung from pneumonic consolidation is very difficult. I cannot state definitely any means by which I can tell the one from the other; but the fact that they suddenly get well without showing those signs of resolution which the pneumonic cases show, and the fact



Temperature, black lines; frequency of whooping, dotted lines.

CHART 1.—Showing the diminution in frequency of whoops when the temperature rises and increase when the temperature falls.

in the frequency of whoops, and also in their severity.

Another point which we have noticed has been this: Occasionally cases are admitted which are bad, with a temperature of 102° or 103°, with signs in the chest which would justify the diagnosis of consolidation from pneumonia. After the child has been in the ward twenty-four to forty-eight hours the temperature falls and the number of whoops is lessened, and one sometimes finds the house physician adopting an apologetic attitude for that the dulness clears up very quickly, indicate the nature of the case. In young children collapse of the lung is, in my experience, attended with rise of temperature. Be on your guard before making the diagnosis of pneumonia simply on account of the temperature and physical signs in a case of whooping-cough.

Another point is the very marked improvement which children show when they are taken into the hospital. A case may be very bad outside, and you take it in, but after it has been in a little while

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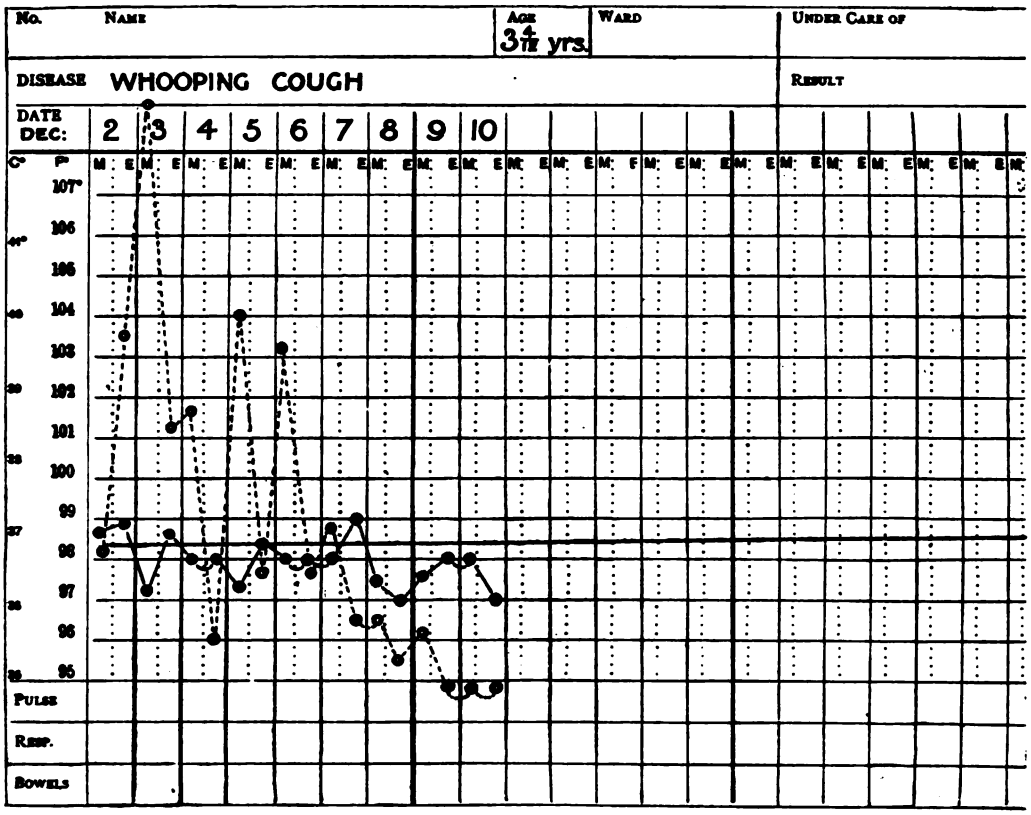
you cannot observe any phenomena which are particularly interesting. I show you here a chart (No. 2) which indicates the beneficial effect of "masterly inactivity" in the matter of treatment. You can imagine well from that that, as a rule, we hesitate before attributing success to any given treatment by means of drugs in whooping-cough until the cases have been watched some little time.

COMPLICATIONS.—Now, with regard to the complications of whooping-cough, I have pointed out that the majority of cases are admitted with some complication already present. By far the most frequent complication is broncho-pneumonia. Out of 161 cases admitted we had fifty-six in which either broncho-pneumonia or lobar pneumonia was, in our opinion, present. The gravity of this condition may be imagined from the fact that twenty-eight, or 50 per cent. of them, proved fatal. Bronchitis was a marked feature in eighteen cases, but of these only one terminated fatally. Pulmonary tuberculosis, usually associated with wide-spread tuberculosis, was present in fifteen cases. When I say it was present I mean that it was recognised as being present. From what one knows of post-mortem examinations one feels it was probably present in more than that number; but we knew it to be present in fifteen cases, and of these fifteen, fourteen proved fatal. Bronchiectasis was present in two, empyema was present in only three cases, and one of them proved fatal. Serous pleural effusion was present in three cases, and all of them recovered.



Temperature, black lines; frequency of whooping, dotted lines.

CHART 2.—Showing the improvement due to good nursing and hygiene. No drugs administered.

Marked distension of the lung, which is often spoken of as acute emphysema, though this is not a good name, was present in two cases in sufficient degree to constitute a marked feature of the case, although it is present in a large number of cases as a temporary condition. So-called surgical or subcutaneous emphysema was present in two cases, both of which terminated fatally. One was in a

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case of extensive tubercular disease in a child. The child had tubercle in most of the organs, some old intestinal tubercular ulcers, and a perforating ulcer of the stomach. I did not see the autopsy on that case myself, but I should think, from what one has seen of tuberculosis, that this must have been a tubercular ulcer of the stomach, which perforated and set up purulent peritonitis. In this case there was an extensive surgical emphysema. Tetany was present in four cases, one of which proved fatal long after the condition of tetany had been recovered from, as a result of an attack of broncho-pneumonia. Another little point which is not incidental to whooping-cough, but I think is an important little practical point, is that ischio-rectal or gluteal abscess occurred in five cases, and from their occurrence we have learnt to look out for them as a cause of a rise of temperature which we could not explain in any other way. Under the head of convulsions I do not include those which were symptomatic of tubercular or non-tubercular meningitis or other actual organic disease of the brain. Convulsions were present in nine cases, and in six of these death occurred. Epistaxis was present in five, hæmoptysis in ten, three of which were fatal, otorrhœa in four cases, and we had one case in which aphasia, the result probably of cerebral thrombosis, occurred.

The mortality from whooping-cough in my ward has been very high, namely, 28·5 per cent. That is to say, out of 161 cases there were forty-six fatal ones. Of these forty-six, thirteen occurred in children under one year of age, and eighteen under two years of age. That means three quarters of the fatal cases were in children under two years of age. Broncho-pneumonia and tuberculosis account for forty-two out of forty-six fatal cases.

To give you some idea as to the relative ages of children who are admitted to the ward, I may say that out of 161, thirty-two were children under one year, thirty-four were between one and two; twenty-seven between two and three; twenty-nine between three and four; seventeen between four and five, eleven between five and six; ten between six and twelve. So you see the vast majority of cases occurred in the first four years of life. With regard to the sex, there is no marked difference beyond that difference which has always been exemplified by the question why the white sheep eat more than the black ones.

TREATMENT.—This is a part of the subject which one cannot but help approaching with a slightly apologetic attitude. Some time ago I was looking up the drugs which had been used in the treatment of whooping-cough. I found a list bigger than I could find for any other curable or incurable disease. When you find a large number of drugs which have the reputation for being more or less specifics for a disease, you can be quite sure that no single one of them is of much good. You will perhaps appreciate the difficulties which one has in some of these cases in judging of the effects of drugs when I remind you that some of these patients scarcely whoop at all, some have only a slight cough, and in others the complications of the disease which are present make one averse to undertaking any experimental therapeutics. The drugs which I have used have been antipyrin, belladonna, citrophen, bromoform, heroin, glycerine of carbolic acid, bromide of potash and belladonna, creasote, chloride of ammonium, carbolic spray, thyroid extract—and I need not apologise for using thyroid extract, because we had a child with malignant disease of the kidney, and we thought we would give it thyroid extract in the hope of influencing the new growth, but it had no effect in diminishing the whoop. Lately we have been going back to a very old practice, namely, the use of antimony. A Swedish doctor who was over here recently told me he knew of a specific which was much used abroad, and asked me to try it. Its composition is Antimon. Sulphurat. rub. gr. $\frac{1}{6}$, Extract. Hyoscyami gr. $\frac{1}{4}$, Pulv. Glycyrrh. gr. $\frac{1}{4}$, Pulv. Gum Acac. gr. $2\frac{1}{2}$, Pulv. Sacch. Alb. ad gr. x. We tried it, and one case has done well on it, but further experience has not impressed me with its value. Besides those we have tried more or less generally carbonate of ammonium, strychnine, phenacetin, guaiacol, salol, and so on. I propose to summarise shortly what our experience has been.

Antipyrin.—This was tried in eleven cases. There was distinct improvement following its administration in two of them. In three there was slight improvement; and in six it was doubtful if the drug had any good effect.

Belladonna.—This drug was given in seven

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cases, and in three decided improvement took place. In two the patients seemed to improve; in two the effect was doubtful.

Citrophen was used in ten cases, in five distinct improvement took place, in one there was slight improvement, and in four there was little or no benefit. In one very obstinate and severe case in which bromoform for two weeks, creasote for two weeks, antipyrin for one week, and glycerine of carbolic acid for three weeks had all failed to give relief, citrophen did undoubted good, though it is only fair to remember that this was only after seven weeks' stay and treatment in the hospital.

Bromoform was used in eleven cases. Its use is not unattended with danger on account of its oily nature and high specific gravity. We have had one case of an out-patient admitted with symptoms of acute bromoform poisoning, and one case has occurred in the ward where the intoxication was mild; in both cases the symptoms followed shortly after the administration of the *last* dose in the bottle.

Bromoform should be dispensed in a dark bottle kept in the dark, and the quantity of the mixture in the bottle should not be too great, so that the risk of an overdose of the drug in the later doses is diminished. Directions should be given that the bottle must be well shaken each time before pouring out the draught.

The dose we have employed has been from 3 to 5 minims three to four times a day.

In two cases there was distinct, in two cases slight, and in seven cases no definite improvement.

Glycerine of carbolic acid was used in fifteen cases; in one case there was distinct, in three slight, and in eleven there was no appreciable benefit traceable to its use.

Creasote was tried in seven cases; in two there was distinct, in one slight, and in four no definite improvement. The drug was given internally and as a vapour. One objection to the use of the vapour was the difficulty of confining the effect of the creasote to a given case for the whole ward, and the nursing staff in the ward became redolent of creasote.

Chloride of ammonium was tried in the hope that the drug might cause the secretion to be less viscid, and so easier of expulsion. The relief to a paroxysm of coughing which follows the mechanical removal of the viscid mucus from the fauces and mouth of a child suggested its employment, but the results obtained with it were by no means satisfactory. I employed it in four cases; in one it gave relief, but in three it was useless.

Heroin was only tried in two of the cases I have analysed for you to-day, though I am giving it a more extended trial at present. In one of these cases when pushed to doses of one thirtieth of a grain every six hours it was very beneficial. In the other case it was of no benefit.

Carbolic acid spray was used in four cases with slight improvement in two, and none in the other two.

Bromide of potash and tincture of belladonna were tried in six cases; one benefited distinctly, two slightly, and in three there was no improvement.

From this *résumé* of the results of treatment by certain drugs I am afraid that I cannot claim to have discovered or even confirmed the existence of any drug which could be called a specific for the disease.

Antipyrin, belladonna, and citrophen have given on the whole the most satisfactory results, though none of them has proved sufficiently beneficial to entitle it to exclusive use.

Creasote and carbolic acid are useful where there is much gastric and intestinal trouble.

Heroin certainly relieves the paroxysms of coughing.

During convalescence nothing has proved so useful as cod-liver oil and steel wine.

WITH DR. E. W. AINLEY WALKER AT GUY'S HOSPITAL.

General Pathology of Inflammation, IV.

Besides the reaction which affects the leucocytes of the blood-vascular system, there are also certain changes which occur in the cells of the tissues involved in the results of the injury which has caused inflammation.

Scattered throughout the tissue spaces are numerous so-called wandering cells. These present many points of similarity to the white corpuscles of the blood, and they belong to the same

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great cell group, forming a part of the cœlomic class of leucocytes. These are probably the leucocytes which can be seen accumulating in the area of inflammation even before diapedesis has become well marked, and which in later stages occupy the boundary zone enclosing the accumulated mass of polymorphonuclear leucocytes in the central area.

The fixed cells of the tissues also undergo important changes in the course of inflammation, and Virchow, as we have seen, attributed the whole reaction to an increased metabolism or over-action on the part of the tissue cells. Accordingly he and his followers ascribed the origin of pus corpuscles wholly or partly to a proliferation of fixed tissue cells which they believed took on amœboid movement.

In support of this position numerous experiments were made by Hoffman and Recklinghausen among others. These observers produced corneal inflammations and then removed the eyes or the whole heads of the animals in question and preserved them in moist chambers. After the lapse of two or three days from this procedure the site of injury invariably showed a great accumulation of amœboid cells, exactly similar to those seen in ordinary keratatis. These they concluded must have arisen from the corneal corpuscles. The fallacy in this experiment is that the cornea remains attached in the normal manner to the eyeball, which itself in some of the experiments was not even separated from the head; conditions which leave it equally possible that all the amœboid cells seen in the area of inflammation had migrated to it under chemotactic influence from the tissue spaces and vessels of the eye, and were not formed at all from the fixed tissue cells.

That this is in reality the fact, and that all these amœboid cells were actually leucocytes, is proved by an experiment of Leber. Leber *excised* the cornea of an animal and introduced into it a tiny capillary tube containing sterile septic material. The cornea was then placed within the peritoneal cavity of another animal. On subsequent examination it exhibited round the focus of irritation introduced in the capillary tube a great accumulation of leucocytes which had invaded it from the peritoneal fluid in which it had been lying, and thus produced appearances precisely similar to those obtained by Recklinghausen.

The views of von Recklinghausen were carried still further by the statement of Rindfleisch and others that *epithelial* cells can give rise to an endogenous formation of amœboid cells within their substance, and these can actually be seen lying within the epithelial cells. These we now know are simply leucocytes which have made their way into the bodies of the cells in question. They are similarly to be seen in cancer cells. We shall, therefore, probably be justified in holding that *all* the amœboid cells of acute inflammations are migrated wandering cells.

In the fixed tissue cells themselves two opposed series of changes occur in association with the inflammatory process. These are respectively *degenerative* and *regenerative* changes. Of the degenerative changes the greater number and perhaps all are not reactive, but resultant; that is to say, they are the direct effect of the injury or of the action of the irritant, and as such are not an essential part of inflammation, which is a process of reaction, but merely associated phenomena.

There are, however, certain degenerative processes commencing or associated with proliferation of cells which are regarded by most pathologists as being of inflammatory nature. These are the parenchymatous degenerations, of which a good example is the parenchymatous nephritis induced by certain drugs or in conditions of acute sapræmia.

Cantharidin, for instance, if it be given in excessive doses, may excite an inflammation of the kidney which affects especially that portion of its substance which contains the excretory segments of the uriniferous tubules—namely, the cortex of the organ. This action we explain as follows:—The cantharidin circulating in the blood in great dilution is taken up for excretion by the renal cells in question, and consequently if it be present in the circulating fluid in such quantity as to reach the kidney cells faster than they are capable of excreting it, it becomes gradually heaped up within them, and ultimately acquires a concentration in those cells sufficient to cause injury, and thus give rise to an inflammatory reaction.

In considering the tissue changes which result from the action of irritants two things are clearly to be borne in mind. ***Firstly, that stimulation, if excessive either in degree or in duration, ceases to be stimulation, and becomes irritation or injury; and secondly, that the same irritant,*** acting for the

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same time and in the same degree, *may produce injury in a highly organised tissue, while only causing stimulation of less highly organised cell systems.* The former of these propositions is well illustrated by the action of cantharidin itself, which in moderate doses stimulates the excretory renal cells, increasing their metabolism and acting as a diuretic; though in larger quantities it causes injury by excessive stimulation, and leads to cell degeneration, lessened metabolism, and diminished excretion of urine. The latter is exemplified in the so-called interstitial nephritis, in which the irritant, whatever it be, while causing atrophy and degeneration of the proper kidney tissue, leads to a stimulation and increased formation in the fibrous stroma.

The action, therefore, of an irritant on any given cell depends entirely on the degree of concentration in which it reaches that cell, and the length of time for which it acts upon the cell. This is illustrated in the formation of a corn upon the foot, which, as pointed out by Paget, is the result of intermittent moderate pressure. If the pressure be made not moderate or not intermittent, we have as the result, instead of proliferation and increase of epidermic structures, their degeneration and destruction with the production of a sore, or perhaps the outpouring of inflammatory exudate and the formation of a blister at the injured part.

It is, therefore, reasonable to regard the proliferation associated with parenchymatous inflammations as the effect of stimulation by the irritant while it is still dilute; and the degeneration as the result of the injury produced by its more powerful and more extended action. And it is important to remember in this connection that the proliferation marks the earlier stages chiefly in acute parenchymatous inflammation.

The degenerations which occur are chiefly the cloudy and the fatty changes, and so-called reversionary degeneration; but the mucoid, amyloid, and hyaline forms are also seen, especially in the chronic inflammations.

Repair.

Besides degenerative changes there is associated with inflammation the process of repair. This is a relatively late event in inflammation, and appears to me to mark the stage and be the result of a weakening of the irritant to the point at which it is no longer injurious, but only stimulating in its action. Epithelium is restored by the proliferation of epithelial cells which have survived, and tissue which has been destroyed in mass becomes replaced by fibroblasts, which ultimately develop into fibrous tissue and form a firm cicatrix.

Repair commences at the margin of the inflamed area where the irritant is naturally present in the lowest concentration; and it begins to appear as the inflammation subsides or passes gradually into a chronic stage; that is to say, as the injurious action of the irritant diminishes to the point where its effect is merely stimulating to those cells concerned in the repair. This is well seen in an experiment of Ranvier, who endeavoured to investigate the peritonitis which resulted from the injection into the peritoneal cavity of animals, of weak solutions of nitrate of silver. Such injections gave the following effects: at the end of twenty-four hours there was in the parts which had been most affected an acute inflammation with complete loss of the peritoneal endothelium, but in the parts more sheltered where the irritant had evidently only arrived gradually and in great dilution, the endothelial cells, so far from being lost, showed evidence of enormously increased activity and growth, and within forty-eight hours some of them had attained a diameter of 100 μ ($\mu = \frac{1}{1000}$ mm.) or more, and had become amœboid.

Similarly with regard to fibrous tissue, its formation is induced by stimulating irritant action. This may be shown as follows: an open wound heals usually by granulations, but if a wound be made aseptically and be left open (that is, the sides not brought together), but be preserved from all mechanical or chemical irritation by absolute asepsis and a non-irritating covering, no granulations form. The epithelium gradually spreads over the surface from the edges, and healing occurs with a minimum of new formation of fibrous tissue. The best natural example of this is the healing which occurs beneath a scab.

The origin of the fibroblasts, from which new fibrous tissue is formed, has been the subject of many researches and of much discussion. If newformed granulation tissue be examined with the microscope, four main varieties of cells are seen composing it. Two of these kinds are obviously leucocytes; the third consists of cells of various

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sizes, but mostly large, of varied conformation, but on the whole of spindle shape, and having a single oval nucleus and abundant protoplasm; these are the fibroblasts. The fourth variety is composed of moderately large cells rounded in shape, with a single nucleus, and relatively a large amount of protoplasm. It is the origin and destiny of this fourth class of cells which still remain uncertain. They are apparently large mononuclear leucocytes; do they develop into fibroblasts?

This question of the origin of fibroblasts cannot be answered definitely in the present state of knowledge. There is no doubt that a large number of them are developed from the fixed tissue cells, the connective-tissue corpuscles of the part. It is the tissue cells, whether of epiblastic, hypoblastic, or connective-tissue origin, which present during regeneration of tissue the most marked evidence in their enlargement, karyokinesis, and proliferation. And in the corneal experiments to which I have more than once already referred, the tissue cells complete the process of repair without the occurrence of any leucocytic infiltration, by the enlargement and proliferation of the corneal capsules. And these large cells belonging to the fourth variety seen in granulation tissue are indistinguishable from a stage in the development of fibroblasts from fixed connective-tissue cells.

On the other hand, they are equally indistinguishable from large hyaline leucocytes. And Metchnikoff has actually watched the development day by day of a large hyaline leucocyte into a fibroblast in a tadpole's tail. Hence it is probable that a proportion of the fibroblasts derive their origin from these leucocytes.

Again, the cells of which I am speaking can be seen to act as phagocytes taking up degenerating polymorphonuclear leucocytes. The same is true of fibroblasts according to Nikiforoff, and of the hyaline leucocytes as shown by Metchnikoff, Ruffer, and others. Their phagocytic action, therefore, does not assist us to decide their origin, whether from tissue cells or from the hyaline leucocytes. All that we can at present say with certainty is that fibroblasts are undoubtedly formed by the proliferation of fixed tissue cells, and probably also to some extent from hyaline leucocytes. German pathologists, however, with the exception of Arnold, all at present hold that connective-tissue cells can only arise from pre-existing connective-tissue cells.

In this connection it is important to remember the view which has been more than once put forward, that the hyaline leucocytes differ from other white corpuscles in being derived from endothelial cells. And we have seen already that in inflammation the endothelial cells, both of the vessels and of serous membranes, become amœboid and throw out processes, and act as phagocytes—become, in fact, very similar in appearance to the large hyaline leucocytes. The fibroblasts derived from the fixed tissue cells are also phagocytic. Thus there exists no very sharp distinction between the hyaline leucocytes, the endothelial cells, and the connective-tissue corpuscles. The leucocytes, the endothelial cells, and the connective-tissue cells are all of mesoblastic origin, and form the simplest tissues of the body, so that it is possible that they are partly interchangeable. And Metchnikoff emphasises this relation by his application of the term "fixed phagocytes" to the two latter classes. How close the relation is between the other mesoblastic tissues and the leucocytes is shown by his own observation that in the degeneration of muscle-fibres in the tadpole's tail the proliferated nuclei of muscle-fibres become the nuclei of separate wandering cells.

Besides the production of new fibrous tissue there occurs in the process of repair a formation of new blood-vessels and vascular loops. The observations of Rindfleisch led to the conclusion that this takes place by an arrangement into parallel rows of extra-vascular vaso-formative cells, thus giving rise to new capillaries which acquire attachment to the existing capillaries of the part. According to other observers certain cells of the granulation tissue become hollowed out to form new vascular channels, just as occurs in the vascular area of the chick embryo. Arnold, however, and most recent investigators deny these statements, and describe the formation of new vessels as occurring by the proliferation and projection outwards of endothelial cells of pre-existing capillary loops. There is first formed a protrusion from the endothelial cells. This becomes nucleated, and grows by the proliferation of the cells which form it. Such a protrusion eventually meets a similar protrusion from a neigh-

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bouring loop and fuses with it, and ultimately this solid process becomes hollowed out to form the new capillary.

There is thus formed to replace the tissue which has been destroyed new fibrous tissue richly supplied with blood-capillaries. In this new tissue further changes then proceed. The fibroblasts gradually become more elongated, and break up longitudinally into successive layers of fine fibrillæ, until eventually but little is left of the original cell except the nucleus and a small amount of protoplasm surrounding it. These fibrillæ undergo chemical alteration and proceed to gradual contraction, which results in the occlusion of the majority of the new capillaries, and ultimately there is produced a firm and almost bloodless, hard cicatrix.

This is the process which invariably occurs in the healing of a wound. Where healing is by primary intention the amount of injury and irritation of the tissues is extremely small, and the formation of new fibrous tissue is very slight indeed. In healing by granulations, on the other hand, the new formation is considerable, and increases in amount with increase in the duration and degree of irritation present. Thus it is least in an aseptic wound which is allowed to heal beneath non-irritating coverings, greater in one irritated by pent-up exudation or by irritating dressings, and greater still in one subjected to the action of powerful chemical reagents or attacked by septic processes.

The process of regeneration leads in chronic inflammations where the irritant is just sufficiently powerful to induce continuous degeneration in more highly organised cell systems, but only strong enough to stimulate connective-tissue growth to the production of inflammatory fibrosis and fibrous hyperplasia.

Relation of the Nervous System to Inflammation.

As regards the relation of the central nervous system to the inflammatory process, we have already seen that its action is not essential, since an inflammation can be produced, and indeed is caused more readily, and runs a more rapid course in parts whose nerve supply has been divided than in normal tissues. And Metchnikoff has followed inflammation in the tail of a tadpole, which had been curarised to such a degree that its heart had ceased to beat, a dosage under which all efferent motor impulses must have been completely blocked.

The time relations also are not those which should undoubtedly present themselves if inflammation were dependent on a nervous reflex; for Cohnheim showed that if, for instance, croton oil be rubbed upon a rabbit's ear quite half an hour may elapse before any evidence of inflammation appears, although when it then arises it assumes a most acute and very active type.

Whether the central nervous system can directly modify the liability of a part to inflammation, independently of vascular changes which it may originate, or can control the type of inflammation which arises, is a more difficult question. The existence of special trophic nerves is, at any rate, very problematical, and is certainly not proved. Indeed, the evidence tends in a contrary direction. The tendency of paralysed limbs, and of parts below the site of injury or disease in complete transverse lesions of the spinal cord, or of the eye after removal of the Gasserian ganglion, to rapid and destructive inflammations, affords no evidence of the existence of a trophic action of the nervous system. They are sufficiently explained by the diminished circulation and by the loss or diminution of sensation which result, by which the vitality of the tissue is lowered, and the perception and removal of injurious agencies prevented. The paralysed limb kept warm and protected from pressure, and the eye kept covered to prevent the entrance of foreign particles into the conjunctival sac after removal of the central ganglion, do not become inflamed. The increased liability to inflammation is therefore not directly the result of the removal of accustomed trophic influences.

Nevertheless there are a number of cases which are extremely difficult to explain except on the hypothesis of direct nervous influence. Such are the cases of inflammation affecting the part supplied by one branch of a nerve associated with definite inflammatory phenomena in other areas of distribution of that nerve; the case of so-called sympathetic panophthalmitis; and last and most important the occurrence of herpes zoster, which is probably always secondary to inflammation of the posterior root ganglion of the nerve supplying the affected area of skin.

All this, however, need not necessarily mean

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more than that a variety of changes in the innervation of a part, and in its vascular conditions, may so lower its resistance and vitality that its susceptibility to inflammation becomes sufficiently increased for the ordinary stimuli arising from normal contact with the outside world to excite an inflammatory reaction. And all these cases of associated inflammation occur in exposed parts. The nephritis sometimes following catheterisation or operation on the urethra, which has been quoted as an inflammation of nervous origin, is, of course, always due to septic agencies.

Cases have been recorded of the apparent production in susceptible subjects of a condition of inflammation by hypnotic suggestion. The suggestion to the hypnotised patient that a red-hot iron had touched the hand, was followed by the production of all the local appearances of a burn. Here the inflammatory reaction, if such it be, appears to have been definitely originated by centrifugal nervous impulses, which it would seem had acted in the direction of an injurious local irritation.

However this may be, there is no doubt that the central nervous system exerts a powerful influence in controlling the origin and course of inflammation through the action of the vaso-motor nerves. Thus we have seen that after the division of the nerves supplying a given region, an inflammation which arises in it runs a more rapid course than in the normal animal. Moreover this may be in part the result of actual changes in the vessel walls as well as alterations in their calibre, for it has been shown by Gergens and Rütimeyer in the frog that after destruction of the spinal cord the vessels allow a greater transudation than the normal, and even permit the passage through their walls of solid particles of colouring matter introduced into the circulation.

An illustration of the influence of the vaso-motor nerves on inflammation is seen in the following experiment of Samuel on the rabbit's ear. If the sympathetic nerve supply, *i.e.* the vaso-constrictor fibres, be divided in one ear, and the auricular nerves—the vaso-dilators—in the other, the vessels of the former become widely dilated, those of the latter constricted from the unopposed tonic action of the undivided nerves. If now both ears be plunged in water at 54° C. for a short time and then removed, the reactions they exhibit differ very markedly. The ear in which the vessels are already dilated from the removal of constrictor impulses becomes still more hyperæmic, and acute inflammation supervenes. This rapidly passes on to a complete recovery, while in the ear of which the vessels are constricted no hyperæmia occurs, but stasis is induced, and the condition may go on to gangrene.

These results have been confirmed by Roger in a modification of the experiment in which one ear retained its normal nerve supply, while in the other either the constrictor or the dilator fibres were divided. Both ears were then inoculated with the same dose of the streptococcus of Fehleisen. If the constrictor had been cut the erysipelas resulting from the inoculation appeared more quickly, and ran a more rapid course in the ear concerned than in the normal ear, while in an animal in which the dilator fibres had been severed it developed much more slowly than in the intact ear, ran a more protracted course, and led to loss of tissue from necrosis.

But although the controlling influence of the vaso-motor system is so definitely manifest, we cannot look upon the vascular changes of inflammation as a vaso-motor reflex, since section of all the nerves does not prevent the usual sequence of the vascular events, though it accelerates them by liberating the vessels from the sway of central controlling influences.

Whether the changes are dependent on a peripheral reflex due to a stimulation of the peripheral ganglion in the vessel walls is a more difficult question, and does not at present admit of definite answer or dogmatic statement. But three considerations are of interest in this relation. First, were the dilatation a peripheral reflex occurring in the local ganglion, one would expect that it would overcome the condition of tonic constriction following the section of the dilator nerves in the experiments already quoted, and this is not the case. Secondly, the evidence, so far as it goes, tends on the whole in favour of the view that the peripheral ganglia of the vascular system subserve sensation rather than the supply of motor impulses. And thirdly, there is definite evidence, as shown by Klebs, that the capillary walls, at any rate, are endowed with independent contractility, so that they are capable of altering their calibre in response to stimulation entirely independently of

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nervous influences. And it is possible, though not proved, that the muscular walls of the arterioles possess a similar power of independent regulative action.

Summary.

Inflammation has been divided according to its *most prominent features* into different varieties—serous, fibrinous, suppurative, ulcerative, productive, and the like; according to the *tissue affected* into parenchymatous or interstitial, and according to its *causation* into simple and infective. It has been said to vary with the *nature* of the tissue involved; the *position* of that tissue on a free surface or otherwise, for example; the *nature of the irritant*; and the *intensity of the irritation.*

Some of these distinctions are useful for descriptive purposes and from the clinical aspect, but all are purely artificial. There is only *one kind* of inflammation, and the differences observed in different cases depend exclusively on the extent to which one or another feature of the process becomes prominent. The actual process is the same in all. It is therefore more correct to use the term *degrees* of inflammation, rather than varieties. For all the events observed depend exclusively upon the factors which we have already treated, namely, the degeneration of injured tissue, the exudation, the migration of leucocytes, the formation of fibrin, and the peptonisation caused by the action of bacterial ferments, where such are present, which occur in the reaction of inflammation, and on the associated reaction of regeneration which follows as the inflammatory events subside.

Thus *serous* inflammation is the type where the intensity of the irritation is comparatively slight, giving rise to exudation with the minimum of migration and breaking down of leucocytes, and consequently hardly any formation of fibrin in the clear fluid; or, on the other hand, an inflammation due to a very virulent micro-organism where the leucocytes are rapidly destroyed in the exudate, their substance, together with any fibrin which is formed, being speedily peptonised by the bacterial ferments. In the latter case, however, the fluid resulting is more usually sanious in character.

Fibrinous inflammation may be described as one in which the action of the irritant is more intense than in the case of serous inflammation.

Productive inflammation is the name given where the marked feature is the subsequent new formation of fibrous tissue. Here the irritation is slight but long continued. In *suppurative* inflammation the irritation is both intense in action and prolonged in time. Occurring on a free surface, it is spoken of as *ulcerative* inflammation.

Of the influence of position as affecting both the vascular phenomena, and the amount and character of the exudation (by mechanical conditions), we have already spoken.

Parenchymatous inflammations are such as affect chiefly the essential tissue elements of an organ,—as, for example, the epithelial cells of kidney tubules; while the term *interstitial* is applied in cases where the connective-tissue basis of the organ appears to be primarily affected.

The *way* in which the nature of the irritant and its degree of intensity affect the resulting inflammation has already been discussed. We also saw that if the irritant be of particulate nature the phagocytes will tend to take it up, and to digest and destroy it if it be digestible. Besides this anti-bacterial action of the phagocytes there is developed a bacteriolytic power in the fluid exudate, so that even exudates in which the leucocytes have been destroyed by the infective agent may still possess considerable bacteria-killing power.

Of the *systemic changes* associated with or accompanying local inflammation the great majority are due to the absorption of toxic products from the area of inflammation. These we shall more conveniently consider under the heading of local infection, while inflammatory fever will be included in our discussion of fever in general. Two facts remain to be mentioned: first, that in acute local inflammation, especially when the inflammatory exudation is considerable, a condition of apoplasmia or diminished fluidity of the blood results from the great loss of fluid from the circulation. The resulting increase in the specific gravity of the blood may last for sixty hours or more from the onset of the inflammation, as was shown by Sherrington. The same observer proved that frequently a leucocytopenia, that is a diminution in the percentages of leucocytes, occurs in the early stage of many inflammations. This is not necessarily accounted for, as at first supposed, by a destruction of leucocytes associated with the onset of the inflammatory reaction, but may be

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due to their collection in some area of the circulation, especially in the lungs, as shown by Muir and others. The stage of leucocytopenia is rapidly followed by a marked increase in leucocytic count, which may appear within as little as an hour of the production of the local injury.

The *termination* of inflammation may be by complete *resolution*—a *restitutio ad integram*—which is the most favourable and the most frequent termination. Or it may end in *molar death* of tissue, as distinguished from the molecular destruction which occurs in suppuration, and this is termed *necrosis*. Or thirdly, it may lead to a considerable growth of new connective-tissue, as in so-called *productive* inflammations. On the other hand, it may not terminate at all, but pass into a lasting stage of chronic inflammation.

If now we ask ourselves the question, what is inflammation? we have seen that of the older theories the view of Cohnheim that it is due to molecular changes in the vessel walls must be abandoned; and equally that of Virchow, that it consists essentially in over-activity of tissue metabolism as the result of local irritation.

We have seen that it is the reaction to tissue injury by a *chemical*, that is a not purely mechanical, irritant; and that the tendency of the whole process is to effect the removal and destruction of the irritant concerned. Hence Metchnikoff has described it briefly as the "endeavour" of protoplasm to digest the irritant.

If we review the process in vascular and non-vascular areas in the same animal and in different tissues, or follow it throughout the animal series, we are met by one and only one invariable event, which is the reaction of the phagocytic cells. This, and this only, is the constant character and sign of the inflammatory process.

Avoiding definition, we may therefore summarise the phenomena of inflammation as *the process which occurs in living tissue when it is injured by the action of an irritant, and which invariably tends to the removal and destruction of that irritant.*

THE unique specimen of a dermoid occupying the angle between the ileum and the cæcum referred to by Mr. Bland-Sutton in the issue of the CLINICAL JOURNAL of October 22nd was erroneously ascribed to Mr. C. Hall. This important and interesting specimen was obtained by Dr. Arthur Hall, Professor of Pathology at University College, Sheffield, and is preserved in the Pathological Museum of that institution. This information will be of service to those who are working in this direction, and may wish to have an opportunity of examining the specimen.

HEATH'S PRACTICAL ANATOMY: a Manual of Dissections, edited by J. Ernest Lane, F.R.C.S., Surgeon and Lecturer on Anatomy at St. Mary's Hospital; Examiner in Anatomy for the Fellowship of the Royal College of Surgeons. Ninth edition. London: J. and A. Churchill, 7, Great Marlborough Street, 1902. (With 321 engravings on wood, of which thirty-two are coloured. 12*s.* 6*d.*)

In this ninth edition of Heath's Practical Anatomy, Mr. Ernest Lane has performed his part as Editor so well that after perusing the book the critic has to acknowledge his inability to discover any unnecessary additions. All the important facts in human anatomy are extremely well discussed, and in the description of details the requisite amount of emphasis is imparted with a literary discretion that is remarkable by its absence in some works on human anatomy. Mr. Ernest Lane, in the preface to this volume, says that the original object of the author, Mr. Christopher Heath, has been carefully borne in mind, viz. to provide the student with an accurate and not too elaborate guide for use in the dissecting room, and for purposes of subsequent reference.

The object thus set forth has been most successfully achieved, and the publication has the very rare merit of not containing one page too many. The dimensions of the work have been curtailed without impairing its utility, and this accounts for the omission in this edition of the many references to surgery present in the last. A further improvement will be noted in regard to the placing of the coloured plates, which have now been interpolated through the volume in the descriptions of the regions to which they refer. The book has not been overdone with illustrations, and a word of praise must be awarded to the coloured plates. The method followed in this work is undoubtedly best calculated to teach the student the anatomical groundwork necessary for sound surgery. The admiration of all experienced teachers of anatomy will be aroused by the skilful manner in which the necessary amount of diagrammatic character has been given in those illustrations where clearness of effect is as important as mere slavish fidelity to superfluous detail. The marked success that has attended this publication is due to the fact that the author has recognised that the practical study of anatomy must be founded on a rational basis, and must be characterised in the very highest degree by simplification of the most complex problems.

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A CLINICAL LECTURE ON FLAT-FOOT.

Delivered at St. Bartholomew's Hospital.

By W. BRUCE CLARKE, M.A., M.B., F.R.C.S.,

Surgeon in Charge of the Orthopædic Department.

GENTLEMEN,—To-day, as you already know, I propose to refer to the subject of flat-foot. I have some lantern slides to show you, and, as I have before pointed out, it is more convenient to show these to start with, so that we may dismiss them and have daylight. I will therefore first direct your attention to a few anatomical facts.

To begin with, you will notice that this foot which I show you is in a position of rest; it is the position the foot takes when one goes to bed and lies flat, or puts one's foot on the sofa. This indicates the range of movement of the foot, and it is very important that you should properly realise what that range of movement is. It is slightly different from that which I am accustomed to refer to as a rule. I always speak of the range of movement as 30° in one direction and 60° in the other. Here it is marked 35° and 55° respectively. There is very little difference, though perhaps the figures given here are a little more correct, but the numbers 30 and 60 are easier to remember. Next I want to draw your attention to the arches of the foot, and to the way in which the arches change from time to time as the foot alters its position. These are the features of an ordinary healthy foot. This is the longitudinal arch, and here is a foot a little more arched when the heel is raised and the person stands on tiptoe. In this first slide the tendon of the tibialis anticus is almost in a straight position, in the second it is more or less curved. These slides display, to a certain extent, the way in which the foot alters its shape when one raises oneself on tiptoe. You will notice—and that is the point of importance—the way in which the scaphoid gradually pushes itself under the head of

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the astragalus as the position of tiptoe is reached. That is the point which we shall have to centre our attention upon when we are considering the question of flat-foot, namely, the movements which take place at the mid-tarsal joint, or transverse tarsal joint as it is often called. We have to bear in mind what we understand by the mid-tarsal joint. By this term we mean the joint which exists between the astragalus and the os calcis on the one hand, and the rest of the bones of the foot on the other. That mid-tarsal joint is not half as much referred to in anatomical works as it should be; at any rate, it is of much greater surgical importance than you have any idea of when you are merely studying anatomy. These slides show you generally the ligaments of the toes, the metatarsus, and so on. This is a view of the outside of the foot, and I will ask you just to refresh your anatomical memories. Look at the head of the astragalus, and notice how much it comes down in the case of flat-foot. Here is a section taken through the foot showing the mid-tarsal joint, and the inferior calcaneo-scaphoid ligament in section. This ligament runs from the under surface of the calcaneum to the scaphoid. The astragalus rests in a cradle which is formed by the inferior calcaneo-scaphoid ligament, and the long and short calcaneo-cuboid ligaments act as tie bars and keep up the arch of the foot. Notice the muscles of the leg, the outer or peroneal group, the peroneus longus and the peroneus brevis particularly, because they are the muscles which pull up the outer side of the foot into the position which it assumes in very bad cases of flat-foot, and make the patient walk on the inner side of his foot. Here are some casts from the Museum, which illustrate the same point. This slide shows the muscles on the inside of the foot, and here are the set of muscles which are very important in helping to draw up the arch of the foot; they are the opponents of the set of muscles we have just seen, viz. the peronei. The muscles which pull the foot into the varus position are the tibialis anticus, and the extensor longus digitorum and pollicis; and here is another muscle that helps them—the tibialis posticus.

I now want to draw your attention to the diagrams which represent, from a surgical point of view, the longitudinal and transverse arches of the foot. The transverse arches of the foot are taken across the cuboid and cuneiform bones.

I have only spoken here of two arches of the foot, the longitudinal and the transverse. Sometimes it is customary to refer to three arches,—that is to say, to speak of the longitudinal arch as consisting of two separate portions, one extending along the outer side of the foot and another portion along the inner. In other words, we refer to the foot as if it were a tripod, and speak of it as if it rested on three points only—the great toe, little toe, and heel. But that is more or less a convenient method of description. The main thing we have to recollect is that there are longitudinal and transverse arches, and that the longitudinal arch is the one on which we shall centre our remarks to-day.

What is flat-foot? In one sense you might just as well ask what is disease. I should answer, of course, that disease is a deviation from that normal condition which we describe as health. I think the best answer I can possibly give you as to what flat-foot is, is to show you a well-marked instance of the condition. If you get a definite idea of the type of any condition or disease, and if you then critically look at the various kinds of disease analogous to it, you will readily understand that one person will call a condition disease which another person does not. Therefore, when I want to explain to you what I mean by flat-foot, let me say I mean the giving way of this longitudinal arch of the foot, so that the foot is no longer arched in the position which we see here, but is practically flat, or, in other words, that pathological condition in which the astragalus comes right down on to the surface of the ground. That, I think, is the real definition of what we mean by absolute typical flat-foot. There are many people who are called flat-footed who are not, in the ordinary pathological sense of the term, suffering from flat-foot; that is to say, one person has a foot with a low arch, and another person has a high arch, but both walk equally well.

Flat-foot is divided into two great varieties—congenital and acquired. I shall not say very much about the congenital form, because it is not a matter of very great importance. In the next place, that form is exceedingly rare, and in a very large number of instances is completely recovered from without any plan of treatment whatsoever. I do not think the term congenital flat-foot is at all a good one, and I do not think we should speak

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of congenital flat-foot at all, except in the very rarest cases. Most of them are really instances of slight flat-foot occurring in early childhood.

When we come to the acquired variety we find it is very common; indeed, it is amongst the commonest of deformities. Here is a case of the flat-foot; it is as typical a one as you could want to see. If you want to test for yourselves whether a foot is flat, one of the simplest ways is to get the patient, as I often do in the out-patient room, to dip his foot into a basin of water and then walk across a piece of kamptulicon, and if one foot is flat and the other not, you will notice a great difference in the prints of the two feet.

What are the bony deformities which accompany it? They are, first of all, a tumbling down of the astragalus; to be more accurate, of the head of the astragalus. Later on follows deformity of the scaphoid and cuboid bones. You will find all sorts of statements in books as to what are the deformities which you are to expect to meet with. The deformities will depend entirely on the length of time that the person with the flat-foot happens to have been in that condition. In the early stage the first thing that happens is the coming down of the head of the astragalus. After that, particularly as most of the cases of flat-foot begin before growth is completed, you will very often get a thinning out of the scaphoid. Sometimes the scaphoid is pushed up on to the dorsum of the foot, and when the foot is thus deformed you get extra pressure upon the cuboid, and it gets narrower at its outer extremity, so that in the very extreme cases you get the base of the metatarsal bone of the little toe touching the os calcis. That is very rare, but it does occur in certain cases which are exceedingly well marked and of long duration. Later on you sometimes get ankylosis of some of the bones, occasionally a bony ankylosis between the os calcis and the cuboid, or possibly an ankylosis between the astragalus and the os calcis. If you want to find good illustrations of any of these forms you should look in the 'Progrès Médical' at a paper by Chaput, I think in 1886. There are in that paper some excellent illustrations of the way in which the bones are deformed and ankylosed in extreme cases of flat-foot.

Next, what are the causes of flat-foot? The ordinary causes are spoken of as rickets, rheumatism, weakness of various kinds, and traumatic causes. Well, now, there are four principal causes, and probably you will find plenty of others mentioned, especially rarer ones; but what we have to do with are the ordinary typical causes of the condition.

First of all with regard to rickets. It seems to me that rickets only acts in this way, that it throws the weight of the body on to the wrong portion of the foot, and in that case, of course, it predisposes no doubt to the occurrence of flat-foot, and inasmuch as in rickets you have deficiency of lime salts in the bones, and the bones are softer, they are no doubt more prone to yield to strain. It is in these senses rickets plays a certain part in the production of flat-foot. *In my own experience I can only say that I do not think there is any cause which is so likely to produce flat-foot as rheumatism.* In fact, I am certain that in the vast majority of cases which we meet with, rheumatism is practically at the bottom of all this trouble. You may say, what evidence have you that rheumatism is at the bottom of it? I will relate to you the sort of history which one meets with in flat-foot; that is to say, the typical history of a case of flat-foot which is associated with rheumatism. In one case which I am thinking of a patient related the following history. The patient was a woman, and she said she had had various attacks of pain about her from time to time, which were obviously rheumatic pains. She had had two attacks of ordinary rheumatic fever, and subsequently had had severe pain about different portions of her foot. What was more, she found that this pain was very much relieved from time to time by the same remedies which have relieved her pains in other parts of the body. So much is this the case that I almost as a routine treatment put such patients upon salicylate of soda, because I find it does a vast amount of good. I do not say that every case of flat-foot is benefited by salicylate of soda, but the majority of them are, and the pain of flat-foot almost entirely vanishes under treatment with salicylate of sodium or other analogous drugs. Under the head of rheumatism we must include gonorrhœal rheumatism. Gonorrhœal rheumatism is very rarely the cause of severe attacks of flat-foot, but when it does occur as a cause, it produces some of the very worst and most intractable flat-feet that we come across.

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Weakness as a cause of flat-foot.—You will find it laid down in the books that it is people with weak fibre, and so on, who are suffering from flat-foot, and there are vague statements about weakness having been the cause. Then we hear of people standing too long in the pursuit of their occupations, and so on. It always seems to me that there is but the slenderest evidence to show that weakness or lack of fibre, or whatever term you give it, is, strictly speaking, a cause of flat-foot at all. I do not believe flat-foot is produced in ordinary weak people unless they have some definite disease in connection with their feet. I am certain that the vast majority of these people who are supposed to be suffering from weakness merely, are actually suffering at the time when their flat-foot lesion begins from some form of rheumatic trouble. One of the best arguments against mere weakness producing flatness of the foot is that you may find a person's foot is very much weakened by tubercular mischief, and yet they will not necessarily get flat-foot. You will get flat-foot coming on, or attacks of pain which result in flat-foot, coming on from time to time and being arrested, and then, after a dormant period, they return again, yet the occupation remains the same. You will also find that a great many of the people who are supposed to be suffering from flat-foot in consequence of weakness do so because they have either badly fitting boots and shoes, or else because they have boots and shoes which let in a vast amount of water, and the consequence is that they suffer from rheumatism in the feet.

One of the causes which I do not think is half sufficiently laid stress upon is ordinary hallux valgus, or the painful great-toe joint of adolescence. I draw your attention to one fact in connection with this disease, namely, that nearly all such cases show considerable thickening about the great-toe joint. Always look to see if this joint is affected in cases of flat-foot. For instance, the foot from which this cast was taken has almost certainly been the seat of hallux dolorosus, or painful great-toe joint. Let us think for one moment why hallux dolorosus produces flat-foot, and then we shall see why it is that various traumatic causes also produce flat-foot. In the ordinary way when we walk, that is to say in the natural method of walking, one raises oneself to a certain extent upon one's toes each time one moves forward. By so doing one exercises the muscles, ligaments, etc., of the soles of the feet, as well as the muscles of the leg. But if one has a painful great-toe joint one cannot do this because of the pain which is caused, and therefore one gets into a vicious way of walking. Instead of raising oneself up on tiptoe each step one takes, one walks with the foot stiff and with the feet turned out. That is a posture which is very characteristic of the agricultural labourer when he follows the plough wearing an exceedingly stiff pair of boots. If one goes on walking with one's foot absolutely stiff in that position it is certain, sooner or later, to result in a certain degree of flat-foot, because the tissues will have become weak from lack of exercise.

Lastly we come to the traumatic causes of flat-foot, and amongst those I suppose the most common are dislocations and injuries about the bones of the foot in the neighbourhood of the ankle-joint itself, and also we must include Potts' fracture. Now those deformities cause flat-foot exactly in the same way as hallux valgus does. For a considerable length of time the patient who is suffering from these lesions is unable to use his foot in the ordinary way, and the foot is weakened by lack of exercise. That brings me to one of the indications for treatment which I may refer to here. Whenever you have got a person whose foot is so far damaged that he is unable to use that foot satisfactorily, always rest it moderately and take care of it until he can begin to resume his muscular action; otherwise you are very liable to get flat-foot resulting. Recollect that any injuries to the foot whatsoever may result in flat-foot,—nay more, certainly will produce it if the foot is not exercised.

Next we come to the consideration of the symptoms and signs of this disease. The only symptom that I know which is absolutely unequivocal is the pain. You may naturally say anybody may have pain. Quite true, but if a person complains of a certain amount of pain in or about the centre of the arch of the foot, and if that pain runs down into the great toe or into the heel, always be very suspicious as to whether it is not a condition of incipient flat-foot. In fact, those very pains are, in many instances, nothing more or less than the first onset of a slight attack of rheumatism. Over and over again I have seen patients coming complaining of attacks of pain of this nature. If I treat them with salicylate of sodium they go on for a considerable

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time without suffering any more pain, and may even be able to do without the special boot which has probably been ordered for them before I have seen them. Then perhaps these patients will come back again, and say the foot is troubling them again. On examining the foot you will find there is not much difference, it is painful on one occasion but not on another. Therefore, whenever you have a person who is suffering from pain in the foot, have a good look at the foot first of all, to try to make out where that pain is, and what the nature of it is. If it is rheumatic you will know what to do.

As to the signs of flat-foot. The signs of flat-foot are the tumbling down of the head of the astragalus which I have mentioned. If you ask me what are the other signs of flat-foot, I repeat what I have already said. Look at the various forms of flat-foot, and at the casts which I have in front of me. You may say, Why do you call this first cast which you show us flat-foot when the foot is obviously not particularly flat, when at the same time you speak of this one as not flat-foot when it has such a low arch? The difference between the two cases I have no doubt is—though I know nothing about the history of the cases from which the casts were taken—that in one the patient has held his toes down in this position in which you see them simply because it was painful for him to get them into any other. Once get those toes up, and you would find that the sole of the foot would go down on to the surface of the ground. Even now the bones that form the centre of the arch are obviously out of position. In the second case, though the arch is a very flat one, there is no bony displacement whatever. When the disease is more marked you get eversion of the foot owing to the contraction of the peronei muscles, and sometimes you may get the extreme condition that you see in this third specimen, in which the arch is practically reversed. Here, then, we have a progressive series in which the deformity is gradually increasing, owing to the tumbling down of the arch of the foot. Bear in mind that this deformity to a large extent depends on the amount which the patient walks in the early stages of the disease, and that is why rest and careful treatment is a matter of so much importance.

What is the course which the disease tends to take? (that is one of the most important points), supposing that nothing whatever is done for the patient. I think we may say that in flat-foot there is absolutely no tendency whatever to spontaneous cure except in children, that is to say, that this disease gradually progresses, and gets worse and worse unless you do something for it. Note that in the course of the disease, one of the most important things is the fact that the *pain* appears and disappears from time to time. In other words, the person may have exceedingly painful flat-foot, and may come to you for treatment. You may treat it by any of the various means I am referring to, and the patient may get better, and he may go on with the foot still flat, but not painful, for perhaps six, ten years, or more. And then suddenly there may be another attack of pain, and the foot will tend to get more flat still. If you get these patients at these stages of exacerbation, I do not think you will have much difficulty in satisfying yourselves that, as a general rule, these attacks are due to rheumatism which comes on at different periods of the patient's existence. Sometimes you will notice that a patient increases in weight, and consequently puts a considerably increased weight upon a foot which is already weakened. In some instances that explains why it is that the foot gets worse from time to time, and better again at other times. And I think I may go so far as to say this, that every person who increases inordinately in weight, sooner or later suffers with his feet in one way or another. I am often saying that one of the most important diseases which we have to treat, and which we ought to regard as a disease, is obesity. I do not think we should allow anybody to get beyond a certain stage of fatness without treating him for it. To put five, six, seven, or eight stone more on the feet than they were intended to bear is one of the most fertile causes of an increasing degree of flat-foot that can be imagined; so much is this so that I have been in the habit of saying to some patients with flat-foot, "You are putting too much weight upon your feet; if you take a little of the top hamper off I think your feet will get along better." Perhaps they do not all like it to be put in those words, but I generally find that they take it to heart. They drink a little less beer and eat a little less pastry, and derive a considerable amount of benefit from such a change of dietary. Considerable benefit accrues to their

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digestion at the same time, for the stomach, of course, suffers a good deal from these errors in diet, and any tendency to rheumatism is benefited by such a *régime.*

What are the treatments which we ought to adopt? What can we do for flat-foot in its different stages? The first thing you have to remember is, what is the course of the disease which I have gone over with you? Bear in mind that the treatment which is applicable to one stage of the disease is absolutely inapplicable at another. So long as a patient is suffering merely from pain, I am in the habit of describing it as merely a tendency to flat-foot. If you can get cases at this stage, when there is only a tendency to the condition and it has not yet been established, you will find that you can nearly always cure them by giving them salicylates, simply because the vast majority of these cases at that stage are nothing more nor less than rheumatism. In other words, there is rheumatism in the fibrous tissue of the sole of the foot, and these tissues are becoming gradually softened; the weight of the body acting on these softened tissues causes the arch of the foot to give way. Occasionally you will find instances in which people will tell you that they have had various attacks of rheumatism. I remember one patient whom I watched during three attacks of rheumatism, or, to be more exact, during two attacks in which acute rheumatism supervened, and during one in which the rheumatism was so chronic that the patient could walk about. The pains in the feet were very much worse in the first two attacks than they were in the third illness, and yet during the first two illnesses the patient did not suffer from any lesion in the feet whatever, or from any resulting lesion. But in the third attack he suffered severely. What is the explanation of this? It is a very simple one. In the first two illnesses the patient was so ill that he was obliged to rest in bed, and there was consequently no pressure upon the foot at all; whereas in the third illness the patient was able to walk, and consequently the weight of the body acting on these softened tissues caused a considerable giving way of the arch of the foot.

The next important thing is to train the muscles. If you ask people what sort of treatment they are going to adopt for flat-foot, they will say, in most instances, "muscular exercises." Muscular exercises are all very well, but you must carefully bear in mind that these must not be ordered indiscriminately. I am constantly having patients sent to me at the Orthopædic Department, and I say to them, "What have you been doing?" They say, "I went to Dr. So-and-so, and he told me to do exercises." "Did he show you what exercises to do?" "No." "Did he look at your foot?" "No." That is the way in which very often exercises are ordered. Oftentimes they are ordered for people whose feet are so painful that they cannot do them. Supposing you had a dozen tin tacks in the soles of your feet, and you came to me and said, "What am I to do?" it would be as reasonable for me to say, "Take a little exercise" as it is in many of these flat-foot cases to advise the patients to do exercises. First of all, then, take care that the patient's foot is sufficiently free from pain to enable him to perform exercises. But freedom from pain is not the only condition which is necessary for the performance of the exercises. You may have a comparatively painless foot ankylosed in a certain position, and, if this is the case, exercises will be of no avail whatever until the ankylosed joints are broken down. If the patient thoroughly grasped the situation he would say at once, "I cannot do these exercises at all, I cannot move my feet." Therefore, before you order exercises, always say to the patient, "Let us see what you can do." I get a patient to walk straight up the room, and I begin by saying, "Walk in the way you ordinarily do." Then I make them return on tiptoe. If a patient can walk even five or six yards in the ordinary way, and five or six yards back on tiptoe, he will probably be able to perform such exercises as will be of value for the purpose of treating the flat-foot. But if, on the other hand, the foot is absolutely firmly ankylosed you must break down the adhesions before you advise exercises. You must bring it back to the position in which it is capable of moving. Treat any dislocation which has taken place here much in the same way as you treat any other dislocation; try to reduce it. This is the method which is sometimes spoken of as "wrenching," or by the French as "brisement forcé." To effect it, place the patient under an anæsthetic, rest his heel on the edge of the table, and press firmly on the dorsum of the foot. You will probably both feel and hear the adhesions

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giving way, and will restore the arch of the foot. Having restored it, you will find some difficulty in keeping the foot in that position to which it has been unaccustomed for so long. In other words, having more or less restored the arch of the foot, you have to keep it in position by putting it in plaster for five or six weeks, or longer, as the case may be. At the end of that time the patients will probably be able to perform certain exercises. I say probably, because you must not order the exercises until you are certain that the patients can perform them. The exercises which they ought to perform are, first of all to raise themselves up on tiptoe four or five times night and morning, and then to roll the feet from the outside edge to the inner backwards and forwards, as people do when skating. If they can perform these exercises, I am sure that, provided they get no further rheumatism, they may look forward to, I will not say in all instances to cure of their flat-foot, but, at any rate, to a cure of the symptoms which make this flat-foot uncomfortable to them. If, on the other hand, you find that your patients cannot perform those exercises, in all probability the bones have been much more deformed in the way in which I have referred to earlier in this lecture, and there may be some synostosis between the bones. The result of this is that such patients are unable to get their feet into anything like a natural position. Under those circumstances it is often customary to order supports for them. Personally I am not very fond of supports. It seems to me that ordering supports for people who have feet to walk upon, and feet which are supplied with muscles and ligaments, is much the same as saying to them "You have got to live in this house, but the house is so rotten that it will tumble down unless you have it shored up with scaffolding." What we do is to try to put the house into a good state of repair so as to prevent the necessity of any shoring up, and that is what you should try to do with a flat-foot. Although I do not mean to say that there are never feet which require instruments of various kinds, I say those instruments should be used as rarely as possible. One of the instruments which are used is sometimes spoken of as a flat-foot boot, which may be of the first, or the second, or the third degree, as it is called. Here they are on the table before you. The first is an ordinary simple boot, and a fairly efficient one, but it is one in which the heel has been continued some considerable distance forward underneath the arch of the foot. Sometimes we put into the boots what are called valgus pads, which may be composed of horsehair, or rubber, or leather, or glycerine. Glycerine I do not advise, because it may burst in the boot, and that would not be very comfortable. We also have, for extreme cases of flat-foot, this other form of boot, in which an iron is fixed to the outside of the boot, and a valgus T-strap, which pulls the foot up in this direction. That strap is affixed, directly or indirectly, to the outside iron, and sometimes to a ring that goes round the calf as well. These are the usual methods of instrumental treatment which we adopt for flat-foot. Sometimes we use these steel soles inside the boot. But there is one objection to treatment by all these instruments, namely, that the patients often complain that the pressure on the under surface of the sole of the foot is so great that they cannot stand the instrument. If you want to realise what their feelings are you have only to walk up an ordinary ladder without boots or shoes, and stand with the centre of your instep on one of the rungs of that ladder for ten minutes, and analyse your sensations.

There yet remains the question of operation. And though definite surgical operation is rarely called for, there are a certain number of cases in which it is absolutely essential. There are various operations which I cannot go into at this stage of the lecture,—indeed, I might devote a whole lecture to it. One consists in removing a wedge-shaped piece of bone from the inside of the foot, and so rectifying the deformity by giving the patient a more or less fixed arch to the foot. In the cases which we operate upon the arch of the foot has long been immovable, and therefore by the operation we are not, as is often said, taking away movable joints, but we are giving our patient a foot which he can readily walk upon and which is fixed in proper position in place of one which is fixed and painful, and on which he cannot walk.

Lastly, you may have to perform amputation in certain instances. I have only once performed it for flat-foot, and that was in a man who was a cabinet maker. He had to stand all day at his bench, but the extreme pain from which he suffered prevented his working for more than an hour or two. I put the whole case before him, and, with his consent, I did a Pirogoff's operation for him.

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The result is that he is able to stand at the bench for a long time, and can earn a good living.

I think you will have noticed that the plan of treatment from which you can hope for the best results is the mode which I speak of as muscular exercises, but I ask you to bear in mind that they must be looked at from a sensible point of view. You must look at every foot for yourself, and satisfy yourself that the patient for whom you order exercises is able to perform them. You must also carefully instruct each patient as to the particular exercises he must perform.

There are several cases for you to see, and which I want you to examine. One of them is a case of flat-foot which has resulted from hallux dolorosus; the individual is one of that almost extinct species, the English waiter, he is about forty years of age. There is another case in the incipient stage in a boy æt. 16, who is only beginning to get pain in the centre of the arch of the foot. It is a case which I should describe as a tendency to flat-foot. There is also a third case of a patient who has suffered from flat-foot for about a year, but who in reality, I think, has had rheumatism in his foot for a considerable time longer. At any rate, the deformity is quite characteristic, and I do not think you will have difficulty in arriving at the diagnosis that it is rheumatic, for he has been very much benefited by treatment with salicylates. The other cases will speak for themselves.

Practical Results of the Forensic Serum-diagnosis of Blood.—Uhlenhuth ('Deutsch. Med. Woch.,' Sept. 18th, 1902) has endeavoured to demonstrate the practical value of his method of serum-diagnosis of blood by obtaining from legal sources various specimens of blood, the sources of which were unknown to him. These specimens were tested with known specific sera, and the diagnosis of the origin of the bloods thus made. In all, twenty-one specimens were examined, and in every instance the diagnosis of the origin made by Uhlenhuth corresponded with the known origin as obtained from the legal authorities. Hitherto all of the examinations which have been made along these lines have been so conducted that the examiner himself knew the source of the blood before he made the examination, so that it might be thought possible that his judgment might be influenced thereby.—*Medical Record.*

SYPHILITIC TESTICLE.

By CAMPBELL WILLIAMS, F.R.C.S.

Syphilis affects the testicle in both the congenital and the acquired types of the malady. In the inherited form of the disease it is a most rare manifestation, if one judges from the paucity of recorded cases. Many of these were only discovered post mortem, in the course of a systematic visceral examination, and with the aid of the microscope, the quality and degree of the gummatous infiltration being of such a nature that it would not have been recognisable to tactile sense, even if its presence had been suspected. Perhaps another reason that may account for its apparent rarity is to be found in the absence of a routine practice of examining for a condition which seldom gives external or visible signs to draw attention to its existence. In acquired syphilis the testicles are liable to be affected during the early months following primary infection by a condition known as "syphilitic epididymitis." This exceedingly rare phenomenon may ensue from two to twelve months after the appearance of the initial lesion. It almost invariably starts in the "globus major" or head of the epididymis, and it usually remains restricted to that part of the organ. Occasionally it may involve the greater part of the epididymis by extension; but it stops short of the globus minor, which, together with the body of the testicle, escapes implication. The process consists in the development of an indolent, hard, painless lump in the head of the epididymis, strongly suggestive of a tuberculous nodule. The differential diagnosis of this affection from an epididymitis of an urethral origin consists of an absence or history of a former penile discharge, the position of the nodule in the "globus major," whilst the globus minor, which is the portion primarily affected in gonorrhœa, is not involved. The painless indolent character of the mass, together with its position, might lead one to suspect tubercle, but the presence of other signs, past or present, of early syphilis and the rapid resolution of the nodule under appropriate treatment would prove its true character. Owing to the lack of opportunity for histological examination of an organ affected in

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this manner the pathology of the condition rests on theory and conjecture. Possibly it is due to the effect of the syphilitic toxine, and is of similar nature to the orchitis of mumps, or that which is seen with typhoid, small-pox, or the epididymitis which is presumably of malarial origin.

The ordinary syphilitic sarcocele, which is universally recognised as a tertiary phenomenon, is frequently evolved during what has been termed the "intermediate stage," an indefinite period intervening between the typical secondary and tertiary epochs. The creation of this extra chronological subdivision is the outcome of the impossibility of drawing a hard and fast line of demarcation for descriptive purposes between the two above orthodox phases of the disease. There is great difficulty in saying where one ends or the other begins, in that the symptoms allotted to one period so often overlap or complicate those which are ordinarily considered to be pathognomonic of the other. Testicular implication of a tertiary nature is commonly met with from two to six years after the primary infection. It may even occur during the first year of syphilisation. On the other hand, the gummatous affection may not declare itself until many years after apparent freedom from all signs of the disease. It may form the sole recrudescence of the latent malady, or it may be in addition to other symptoms. It is not a very uncommon sequela at eight or ten years after the initial inoculation. I have recently seen a case of syphilitic sarcocele which supervened thirty-seven years after the primary chancre, the patient having been free, as far as he knew, from all other specific symptoms for thirty-six years. It developed a few weeks after a trivial blow on the testicle, and its true nature was not suspected for some time. In another case the patient developed syphilitic mischief in his right testicle and his left tibia ten years after infection, with a period of apparent freedom of over eight years. This case was most peculiar, for the body of the testicle felt quite unaffected, whilst the whole of the epididymis was involved, so much so that malignancy was at one time suspected. It resolved, however, on large doses of iodide of potash.

Gummatous invasion of the testicle is usually described as tending to be a bilateral rather than an unilateral affection; but it very frequently confines its ravages to a single orchid, possibly because treatment is resorted to immediately the condition is noted. For when the development is symmetrical it commonly appears in one testicle first, and in my experience by preference the left; the fellow organ becoming involved subsequently. I have certainly seen more cases of unilateral invasion amongst private patients, who seek and follow out treatment as soon as they discover something wrong, than with hospital cases, who often allow matters to drift before taking advice, and thus give time for both organs to get affected. In many instances of syphilitic testicle one is able to elicit a distinct and reliable history of former injury to the orchid, which would seem to be the determining cause of the subsequent syphilitic manifestation. This is probably due to the traumatism having impaired the resistance of the tissue by reducing its vitality, and thus rendering it more vulnerable to toxine reaction, if such be the cause of syphilitic manifestations.

The clinical aspect which the gummatous development primarily assumes may be called, for descriptive purposes, "the nodular" and the "diffuse" types. Certain cases, judging by tactile examination alone, would seem to definitely belong to one of the varieties, to the exclusion of the other. This remark is particularly applicable to the smooth sarcocele, where the epididymis is apparently unaffected as far as the sense of touch can make out. But I strongly question whether this seeming freedom from implication in the gummatous process would be verified by microscopical examination if it were possible to employ it. The combination of nodulation and infiltration is of common occurrence, especially when the disease has been allowed to progress untreated. This admixture of the two varieties of gummatous invasion may be present in either comparatively equal degrees, or one of the types may be in marked preponderance to the other. As illustrative of the foregoing statement, one sees cases presenting the following characteristics:

1. Node or nodes of the body of the testicle which tend to increase and coalesce, the epididymis being apparently unaffected.

2. More or less uniform or diffuse infiltration of the body of the testicle, which by its enlargement envelops and obscures a *quasi*-normal epididymis.

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3. A similar condition of the body with nodular excrescences from the tunica albuginea.

4. Infiltration and nodulation of the body of the testicle with nodulation of the epididymis.

5. Nodulation of the epididymis alone, the body of the testicle being soft and seemingly unaffected. This form is the rarest aspect which the gummatous invasion assumes. It strongly mimics a tuberculous deposit. It is needless to point out, excluding the last variety, that the progress and duration of the process is fairly well expressed by the condition discovered, and that, as the invasion proceeds, one is more liable to meet with the combination of sclerosis and nodulation. In the very earliest stage of a syphilitic testicle it may simply appear to be slightly firmer than normal, either uniformly or patchy, the scrotum, cord, and epididymis being, to all appearances, normal and healthy. If allowed to proceed untreated, the organ gradually increases to about twice its ordinary size, becoming as hard as cartilage, and devoid of testicular sensation if pressed upon. This sclerosis affects the rete testis and the sustentacular connective tissue, which, owing to gummatous proliferation, is thickened to an extreme degree. Or it may be that the surface of the testicular body feels uneven and hard in places. If the case be left untreated, these hard points increase in size, forming distinct masses, which tend to coalesce. Eventually the body becomes densely indurated, enlarged to double its normal size, with a tendency to a pyriform shape, so that it overlaps and envelops the epididymis. The contour of the orchid may be comparatively smooth, or, on the other hand, the surface of the tunica albuginea may be studded with nodules. If treatment be not employed, the epididymis becomes involved in a similar fashion. The gummatous excrescences from the tunica albuginea tend to progress, and even implicate the scrotum. This becomes adherent to the adjacent mass or masses. The next step is that the skin ulcerates at the point of union, to give vent to a deeper disintegrating gummatous focus which has succumbed to progressive obliterative endarteritis of its supplying vessels. The resulting ulcer of the scrotum may present the typical chamois-leather appearance, the base reaching down to the outer surface of the tunica albuginea; or the edges may form a circular outlet for a fungating mass of granulation-like tissue which protrudes from the testis through an aperture in its investing fibrous capsule. Involvement of the scrotum is comparatively rare, and a hernia testis is even more uncommon, in that the disease is seldom allowed to reach this stage. I have endeavoured, in the above picture, to trace the progressive steps of an unchecked condition proceeding to the ultimate limit. But fortunately the course of syphilitic disease of the testicle is of slow development, and its ravages are usually arrested before it has made much advance. The sooner specific remedies are resorted to the easier will the resolution be, that is, provided that the remedies are given in efficient dosage, for *quasi*-homeopathic doses are worse than useless in these cases. Vaginal hydrocele of varying degree is very common in connection with syphilitic disease of the testis. The amount of fluid present may be small and trifling, or it may total from twelve to even twenty ounces. One often finds, on tapping a hydrocele, that the concealed testicle is undoubtedly syphilitic, and the patient is even more surprised to hear that his old enemy is the cause of his present trouble, and that he must revert to specific remedies. Under antisyphilitic treatment not only does testicular resolution ensue, but the fluid which in most cases, if not all, tends to keep on re-accumulating after repeated tappings, ceases to do so as the gummatous material is absorbed. It must be borne in mind that the procedure of resolution of a diffuse gummatous testicle is often a tedious business, and requires prolonged and sometimes heroic doses of the iodides. In a case of this nature in the practice of my friend Dr. F. W. Cock, the patient, æt. 50, had taken considerable iodic treatment without any marked effect either upon his testicle or the hydrocele, which went on filling notwithstanding periodical tappings and other local treatment. On the last occasion, after the fluid had been evacuated, the cavity was washed out with an ounce of Liq. Hydrarg. Perchlor., and about two drachms of the solution were left in with a view to exciting adhesive inflammatory action of the opposed serous surfaces. The reaction was great, so much so that at one time it was feared that suppuration would ensue. This, however, did not eventuate, but with the subsidence of the inflammation the hitherto rebellious testicle rapidly resolved, and the hydrocele likewise disappeared.

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It seemed as if the phenomenon was due to the direct local action of the mercury upon the testicle as well as the hydrocele.

The classical diagnostic signs of a syphilitic testicle, above and beyond the alteration of the organ in size, contour, and density, consist in the condition of the spermatic cord, the loss of testicular sensation, the absence of pain, together with the presence of other corroborative signs of syphilis. In the vast majority of cases the spermatic cord is unaffected, being neither enlarged nor thickened. But occasionally the connective tissue entering into its formation is the subject of gummatous hyperplasia. One even meets with a thickened vas deferens in connection with a syphilitic testicle. This is usually the legacy of a former gonorrhœal epididymitis which has left the seminal duct permanently but uniformly thickened, not nodulated as in tubercle. Moreover the vesicular seminalis (which seems to be exempt from syphilitic affection) shows no signs of the latter malady, unless, as rarely happens, the two diseases coexist, a possibility that must be borne in mind. The absence of pain in the testicle itself is a marked feature in syphilis. But this is not absolutely true, as sometimes a certain degree of nocturnal aching is complained of. Such local sensations which are usually experienced consist of a feeling of weight or dragging on the spermatic cord, due to the increased gravity and bulk of the testicle. The uneasiness is often referred to the lumbar or renal region, owing to the anatomical relations of the spermatic artery and its accompanying nerves. Testicular sensation to compression, though slightly increased at the very onset of the invasion, soon becomes diminished, and eventually when infiltration is complete it is entirely absent. Virile power is diminished, and in bad cases lost. This loss may be temporary, and amelioration ensues with testicular resolution. But sometimes it is permanent if both testis have suffered, owing to atrophy of the organs. Although a syphilitic testicle may be the *only* evidence of the activity of the disease, it is more usual to find other confirmatory symptoms in the patient, such as periostitis, periosteal nodes, subcutaneous gummata, a palmar or plantar squamous syphilide, tertiary ulceration of the skin; or perchance a fissured or ulcerated tongue, lips, or fauces tell the tale. On paper it seems quite easy to be positive about the diagnosis of a syphilitic testicle, in fact nothing more simple. Nevertheless in practice it is not always so, for it may be the only sign of a disease of which there is no early history. Patients have been castrated on the assumption that the lesion was either malignant or tuberculous, most frequently the latter, when it has really been syphilitic. Syphilis has been suspected, but because they did not react to specific treatment, and even got worse during the administration, they were operated upon, with the result that the ablated organ showed that the first diagnosis was correct. I well remember the case of a student who escaped this fate. He developed a large node in his left epididymis. He was unaware of ever having had syphilis, though he admitted many possibilities of exposure. His complaint was diagnosed by one of the most scientific of surgeons, a great pathologist, as tubercle, and an operation was advised. Before submitting to this he saw the late Berkeley Hill, who, notwithstanding the history, or, rather, absence of prior syphilitic history, determined to give him the benefit of the doubt, and put him on heavy doses of iodide of potash in lieu of the small ones he had been taking. The result was that the nodule disappeared. The subsequent history of the patient was that he neglected further treatment as soon as the testicular manifestation cleared up, and eventually developed cerebral trouble, which resulted in hemiplegia.

The differential diagnosis of testicular syphilis may be required to eliminate the following conditions, namely, simple orchitis, sarcoma, carcinoma, adenoma, and tubercle. The chronicity of the syphilitic process serves to exclude the acute affections to which the gland is liable. The greatest trouble is usually with early tuberculous or malignant disease. Tubercle primarily affects the epididymis, and although syphilis may do so likewise, it, however, usually selects the body of the testicle to the exclusion of the epididymis. The tendency of a primarily nodular syphilitic testicle is to become more or less smooth. A tuberculous one never does this, but becomes more and more "craggy." The tuberculous orchid is prone to involve the scrotum, to break down, discharge, and form a tuberculous ulcer and sinus. Though a syphilitic gumma may act similarly, it is a much less common event than

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with tubercle. The spermatic cord in syphilis is usually unaffected, and in those rare cases when it is implicated it is the connective-tissue element rather than the duct, whereas in tubercle the vas deferens is usually involved, sooner or later producing the characteristic nodulation. The seminal vesicle, which does not appear to be affected by syphilis, may be found to be involved in tubercle should the infection spread sufficiently upwards. Sometimes, however, tubercle extends from *above* downwards. That is to say, it starts either in the prostate or the seminal reservoirs and descends along the duct to the epididymis. The inguinal lymphatic glands are often implicated in the tuberculous, whereas with the syphilitic process they escape, unless ulceration of the scrotum takes place, and the resulting sore is subjected to irritation. The tuberculous testicle may be tender in its initial stage, but actual pain eventually develops, and increases as the invasion of the organ progresses. It is the opposite with syphilis. The testicle is usually painless, or, at the most, only slightly tender at the onset, and entirely loses this symptom as infiltration proceeds. Tuberculous disease of the testicle is more common during the immediate years following puberty. The liability to invasion decreases with advancing years, although it *may* originate even at middle age. The syphilitic testicle is more frequently met with after the twenty-fifth year of life, because a varying period of years usually elapses between contagion and the orchitic phenomenon. A stethoscopic and general examination, together with a temperature chart, help clinch the diagnosis. The "essential fever of syphilis" is so rare, and has characters of its own, that it would not be confounded with the familiar tuberculous pyrexia. Finally, the presence of a hydrocele would be strongly in favour of syphilis, but it must be remembered that hydrocele is occasionally met with in connection with tubercle.

Malignant disease of the testicle in its *early stage* is liable to be confounded with syphilis. Both sarcoma and carcinoma start in the body of the orchid, but the rapidity of the process as compared with syphilis uncontrolled in the slightest degree by antisyphilitic remedies should quickly rouse suspicion as to the true nature of the complaint. The surface of the body of the testis will be felt to be uneven, in fact, studded with nodules. The individual focal development is rapid, the epididymis and cord soon become involved. Scrotal adhesions occur early, the skin ulcerates, giving vent to the typical readily-bleeding fungating mass; whilst the lymphatic glands of the groin and abdomen become involved in the process at an early period. Pain may be absent at the commencing stage, but ere long it becomes a prominent feature in the case. Malignant disease is usually unilateral, whilst implication of both testicles, though occurring in tubercle, is most strongly in favour of syphilis. Sarcoma may be either of the round-celled variety, commonly the small round-cell, or it may favour the lympho-sarcomatous or the conglomerate adeno-myxo-chondro-fibro-cystic type. Carcinoma is almost invariably of the soft, rapidly growing, encephaloid variety. But it is impossible to be certain as to the exact nature of the malignant growth until the testis has been removed.

The two remaining conditions which have to be considered are adenoma and simple chronic orchitis. The adenomatous tumour (cystic sarcoma, cystic sarcocele) belongs to the *quasi*-malignant type, that is, that though primarily of benign nature it may eventually take on malignant activity. The condition occurs in early adult life. The growth is situated in the substance of the testicle, and increases slowly and painlessly. The diagnosis is much simplified if cystic fluctuation is present. The mass is, likewise uninfluenced by iodic treatment.

Simple chronic orchitis so closely simulates its syphilitic prototype that it is sometimes impossible to differentiate the two conditions. They both yield to similar treatment, but the main guiding points would be—an absence of a former syphilitic history, not that such can always be relied upon, some reasonable and probable explanation of its development, and the unilateral restriction of the process; not that such evidence is conclusive.

In inherited syphilis the testicles are comparatively immune to, anyhow, the grosser gummatous developments, although a few cases have been recorded where even ulceration of the scrotum has occurred, with an accompanying fungating protrusion. Some of the inherited sarcoceles have never given objective or tactile evidence, and have been discovered post mortem. In those cases which have been diagnosed and treated, the enlargement was noted to have been gradual, painless, unilateral, and strictly confined to the body of the testicle. To tactile sense the gland felt

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firm but smooth. The age of the patients when they came under treatment varied from one month to two years, and the condition reacted to anti-specific remedies. Hydrocele was a frequent accompaniment to testicular implication, but it must be remembered that it is quite common to meet with a hydrocele in newly-born infants, who cannot be suspected of being syphilitic. I have purposely tapped some of these for the purpose of examining the testicle, to see whether anything could be felt amiss with the orchid, but have always failed to find any evidence that could be ascribed to syphilis. Moreover, the bulk of infantile hydroceles clear up spontaneously.

The only thing to be said about the treatment of syphilitic sarcocele in the adult is, that if you give iodide give it in sufficiently big doses to effect its purpose; it is no use giving a case of densely sclerosed orchitis a dose of three or five grains of the salt. It is tantamount to wasting time and drugs. Start at least with ten grains freely diluted with water thrice daily, and if that amount does not soon give signs of absorbing the gummatous material, increase to fifteen, twenty, or twenty-five grains three times a day. I have been much impressed by the good results obtained by my friend Professor F. W. Mott, who does not hesitate to prescribe large doses of Pot. Iod. in syphilis of the nervous system, when he deems there is necessity for doing so.

Another point is, are there any circumstances which justify the contravening of the well-known surgical law relative to removal of a syphilitic testicle? I have only once excised a specific orchid. It was done after consultation and at the patient's urgent request. I obtained his written statement to that effect before operating. He had suffered severely from gummatous mischief in the left testicle. On two former occasions it had involved the scrotum and fungated. Twice had he come to England for treatment. His occupation was mainly on horseback, and he, rightly or wrongly, ascribed his past and present trouble to injuring the organ against the pommel of the saddle. On the third occasion he determined to get quit of his local trouble, more particularly since the organ was hopelessly disorganised, and had once more fungated through the scrotum. Consequently he had it removed, and went through a course of mercurial inunction. The patient has been free from further local trouble for many years, and said that he regretted that the testicle had not been removed on a former medical visit to England.

Personally I always prescribe a cetain amount of mercurial treatment subsequent to the iodic regime, as I hold the view that iodide scotches the disease but does not cure, and that the chance of relapse is less if a mercurial course is given in addition and subsequent to testicular resolution.

WITH MR. JAMES BERRY

AT THE

MEDICAL GRADUATES' COLLEGE AND POLYCLINIC.

MARCH 19th, 1902.

GENTLEMEN,—The first case I have to show you is one which you may perhaps call a trivial one, and it need not detain us long. The patient is a boy about ten years old, and for several months he has had pain and swelling in his left ankle, so that he has been unable to walk upon it. You will see, even from a distance, that there is a very obvious swelling in the neighbourhood of the ankle-joint, below, behind, and in front of the internal malleolus. On the outer side there is a similar swelling round the outer malleolus. In front there is a deep-seated swelling below the extensor tendons, and at the back, on either side of the tendo Achillis, there is also a swelling. The swelling is exactly opposite the ankle-joint. When I feel it I find that it is soft and pulpy, but not particularly tender; when I grasp the foot I find that there is considerable lateral movement at the ankle-joint,—that is to say, the whole ankle-joint is loose, and evidently considerably diseased. The diagnosis of the case is that it is undoubtedly one of tuberculous disease of the ankle. Neither can there be any doubt that it has gone on to suppuration. As to treatment, I am afraid it has gone so far that I must cut into it, let the pus out, and drain it antiseptically.

The next patient is an elderly man under the care of my colleague, Dr. Sainsbury, who is kind enough to allow me to show him. He is sixty-four years of age, and he came to the hospital on account of difficulty in swallowing. For the last six months this difficulty in swallowing has increased, both as regards solids and liquids. During that time he has lost a good deal of weight, more than two stones. He has also had a good deal of vomiting, and now whenever he tries to take solid food he vomits immediately, and he also frequently vomits after taking liquids. When I come to examine him I notice first of all that there is a small lump, which is obviously either an omental hernia or a hernia of subperitoneal fat. That has, however, in my opinion, nothing to do with his symptoms. On examining the abdomen I find

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nothing abnormal. One can get one's hand well under the ribs, and no tumour can be felt there. I am told that on examination of the chest nothing abnormal is to be found. When I pass a bougie down the œsophagus I find that it will pass sixteen and a half inches from the teeth, and then it comes to a dead stop. With regard to the diagnosis of this case, it is pretty obvious. I do not think there can be any difference of opinion as to the presence of an obstruction somewhere about the lower part of the œsophagus. Whether the obstruction is actually in the œsophagus or in the stomach is doubtful. As to the nature of it, I am afraid there is no doubt it is carcinomatous, for this is the common cause of loss of flesh and dysphagia. It is rather curious that the bougie should have passed as far as sixteen and a half inches before it met with the obstruction. The distance from the teeth to the œsophagus is normally seven inches, the length of the œsophagus nine inches, so that sixteen and a half inches brings us practically to the cardiac end of the stomach.

[Two specimens of carcinoma of the œsophagus from the Royal Free Hospital Museum were then shown].

I propose now to discuss briefly the treatment of such a case as that which you have just seen. There are three courses open to us with a patient who has epithelioma of the œsophagus. One is to allow him to drink by the natural means as long as he can, to feed him by the rectum, and give morphia to relieve pain. As a rule pain is not very prominent in these cases, and when it occurs it is due to inflammation around the growth rather than to anything else. The next course is to feed him by means of tubes,—that is to say, to pass a tube through the stricture, and then feed the patient by means of that tube. The tube may be passed intermittently every day or two, or it may be kept in. The intermittent passage of tubes in a case of malignant stricture is dangerous on account of the risk of perforation. If you are going to treat a stricture of the œsophagus by means of tubes it is better to put in a short funnel-shaped tube (Symonds's), and let it stay in, changing it once in three or four weeks. The third method of treatment is by gastrostomy. The objection to that is that, as usually performed, it is an operation of considerable danger, and that there are considerable local discomforts. The most serious of those local discomforts is the ulceration and digestion of the skin around the opening. And although that trouble with the skin ought not to occur, it is very difficult to prevent. We find every now and then, in spite of all we can do, that the skin does give trouble, and there is a great deal of pain and irritation. With regard to the passage of tubes, I will show you a comparatively simple method which I adopt usually with carcinoma of the œsophagus. I may say that in the case before us I do not think a tube is at all suitable, on account of the low situation of the stricture. Symonds's tubes are not suitable for a stricture of the œsophagus if the growth is very high up or very low down. For this reason: if it is low down, as soon as the patient vomits the tube is pushed upwards and easily slips out of the stricture; when the growth is very high up, near the cricoid, treatment by the retention of a tube is also open to objection, for the wide end rests against the cricoid and causes ulceration of that part, or, if it is higher up, it causes œdema of the glottis. Therefore, personally, I never use tubes when stricture of the œsophagus is at either end of the digestive tube. But in the case of strictures which are anywhere about the middle of the œsophagus, between the mouth and the stomach, I think the treatment by means of tubes is preferable to that by gastrostomy. But to that there are notable exceptions. If you are going to treat a case of stricture of the œsophagus by means of tubes, there is the very great initial difficulty that you may not be able to get a tube in. In inserting a Symonds's tube I never make use of the stiff instrument invented for the insertion of these tubes. The risk of causing perforation of the œsophagus is so grave that I reject the method. If I want to pass a Symonds's tube, I first of all take a catgut guide or fine catheter, and I attempt, very gently indeed, to get that through. I think the cases where you cannot get anything through an œsophageal stricture are very few if sufficient patience be exercised. After a catgut guide has been passed through, it is an easy matter to pass a catheter over it. It will often be found that a stricture which appears at first wholly impassable will admit a good-sized catheter when once a catgut guide has found its way in. The strictured œsophagus is not only narrow but tortuous. Sometimes you will succeed in passing your Symonds's tube at

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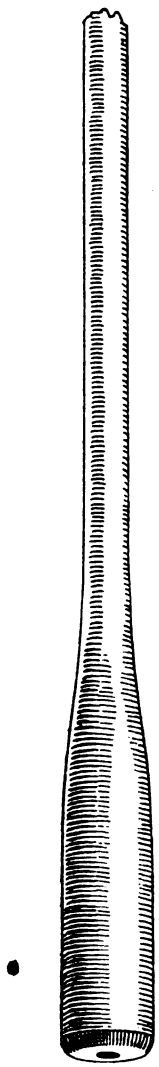
your first visit, but more often it is necessary to have one of these ordinary small railroad gum-elastic catheters kept in the stricture for a few days, until the stricture is sufficiently large to admit the passage of the short Symonds's tube. When you find that the stricture is sufficiently large to allow the passage of the latter it should always be passed on



a guide. Suppose the patient is wearing a railroad catheter (that is, a catheter with a terminal, not a lateral opening), and I want to introduce Symonds's tube. The first thing to do is to pass a long catgut guide down through the catheter. The catheter is then withdrawn, and this Symonds's tube is slipped over it and pushed down into the pharynx with the finger as far as it will go. Then, to get the tube still further down, I use a method which is, I think, not generally known. A longer catheter is used which has its lower end shaped like a bottle (see Fig.), with the broader end downwards. It is passed over the guide, and then by means of that blunt end the Symonds's tube, still on the guide, is gently pushed down through the stricture without any risk of perforation. You must not use force, but if you always employ a guide and carry out the method I have mentioned no force is necessary.

The next two men are both old patients of mine. Both illustrate the surgery of intestinal obstruction. One of them was under my care twelve years ago and the other eleven years ago. Both cases are very similar. This man was thirty years of age, and he was exceedingly ill when he first came to the hospital. For eighteen days he had been constantly vomiting. He had had complete constipation, gradually increasing distension of abdomen, and great abdominal pain. When I first saw him, in a medical ward, he was supposed to be dying of peritonitis. He was much emaciated, the abdomen was extremely distended, he was vomiting and hiccoughing, and had a pulse of 140 to 160, and I was asked to see him with the view of operating, as it was thought to be a case of intestinal obstruction. He appeared to be almost moribund, and it was obvious that any large operation on a patient in his condition would have been fatal at once. A small opening, an inch long, was made into the abdomen, and the first bit of distended gut that presented was sewn to the abdominal wall and then opened. That small operation was very quickly done, and did not appreciably add to the man's symptoms. For a day or two he was exceedingly ill. Then he gradually began to get better, and a large quantity of liquid fæces ran away for a long time, and he had a fæcal fistula. His life was saved, and after that I had to deal with the fistula, and a number of small plastic operations were required to close that. He was in the hospital on and off for about two years at different times, and ultimately he got perfectly well. He was away from his work for two and a half years.

A few months later this other patient came into the hospital with a somewhat similar history, under another physician. In this there was a definite history of a blow in the abdomen a fortnight

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before. He was admitted with peritonitis, and I saw him three weeks later. He was so bad that I would not have him in the theatre, and I operated upon him in his bed, doing the same as in the other case. But in view of the trouble that had occurred with the fistula in the other case I made a smaller opening in the intestine, and after a few weeks I was able to close it completely and permanently at a single operation. The cause of the peritonitis in these two cases I do not know. The physicians who asked me to see the cases were inclined at first to think that there was mechanical obstruction. I think it very unlikely that the cases were of that nature. You must remember that these cases happened eleven or twelve years ago, and I believe that if I saw similar cases now I should think more of the possibility of appendicitis than I did then. I think it very likely that in both those cases there was acute peritonitis resulting from appendicitis. I also believe in regard to both of them that if a large operation, such as opening the abdomen for the removal of the appendix, had been attempted, both of them would have died, because they were so exceedingly ill at the time, and quite unfit to undergo any large operation. They are, to my mind, interesting as cases illustrating what I believe should be the proper treatment of certain very desperate cases of peritonitis in which extreme abdominal distension is the most marked feature.

Preisich and Schütz examined the nail-dirt of sixty-six children with a view to discovering the frequency of the occurrence of tubercle bacilli therein. In fourteen cases, or 21·2 per cent., this was accomplished. The children were not selected from tuberculous surroundings.—*Medical Record*, October 18th, 1902.

Treatment of Vaginismus.—For obstinate cases of vaginismus, Tavel ('Rev. de Chir.,' No. 2) resects the pudic nerve. This is reached on each side by an antero-posterior incision in the corresponding ischia-rectal fossa midway between the tuberosity of the ischium and the outer margin of the anus. The nerve is located by the pulsation of the accompanying artery, and is traced backward, freed from the artery, and divided. The peripheral portion is then twisted out.—*Amer. Journ. of Obstetrics, October*, 1902.

Cancer of the Uterus: a Clinical Monograph on its Diagnosis and Treatment. By Arthur H. W. Lewers, M.D., &c. H. K. Lewis, London.

The object of this book is important, for it has been written to impress upon practitioners that if patients with cancer of the uterus are seen early enough, and adequate operation is undertaken promptly, there is a reasonable hope that their lives may be prolonged and made comfortable for a long period. The author supports this opinion by giving careful details of nineteen cases out of a total of seventy-three in which he has operated for this disease, in which the patients have been known to survive the operation from four to fifteen years without a return of the cancer.

The encouraging tone of this work is very delightful, for in recent years monographs and brochures on the subject of uterine cancer may be termed books of "lamentation and woe," for they are full of wailing in regard to the unsatisfactory results of surgical treatment.

The book is in the main of value for the careful clinical details of the successful cases which Dr. Lewers has followed up with such unusual assiduity. The facts relating to diagnosis are such as are met with in the usual text-books of gynæcology, and the author, by no means anxious to pose as a morbid anatomist, has borrowed somewhat freely in the pathological sections of the book from Cullen, but always with due acknowledgement.

The book shows also that Dr. Lewers, in his early operations, was content to amputate the cervix for cancer in that part of the uterus, but with increasing practice he has, like those operators with the longest experience of vaginal hysterectomy, become a convert to the view that it is in the best interests of the patient to remove the whole organ.

The mortality is perhaps rather higher than usual—four deaths in the forty cases (one to ten), counting the case which died of pneumonia three weeks after operation,—but there can be little doubt that Dr. Lewers will materially diminish this in future lists. We regret that the drawings are so poor. In a few instances they may perhaps illustrate the text, but nowhere in the book can they truthfully be regarded as adornments to its pages.

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* *Specially reported for The Clinical Journal. Revised by the Author.*

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SOME RECENT AND OLD OBSERVATIONS ON GASTRIC ULCER.*

By FRED. J. SMITH, M.D., F.R.C.P.,
Physician to the London Hospital.

GENTLEMEN,—Probably all of us present to-night have been face to face with one of the most distressing and severe accidents that can occur in medical practice, viz. the sudden onset of violent hæmatemesis or perforation of the stomach, so that no excuse is needed for discussing the subject of gastric ulcer, though some might think it was worn threadbare.

PATHOLOGICAL APPEARANCES.—Every one is familiar, both by reading and possibly by actual experience, with the two common forms of definite ulceration of the stomach, *i. e.* the small punched-out so-called peptic or acute, and the large indurated or chronic, but I do not think we have been so alive to what I believe to be a third form. My own experience of this third form has led me to attach very great importance to it. What I am saying will be read by many who are already familiar with the condition, but this account may come before those who have not had any opportunity of noticing it, and I may therefore be permitted to give my experience in some little detail.

The first case that attracted my attention occurred some ten years ago; it was that of a man about forty-five to fifty years of age, who came to me at the City Road Chest Hospital with all the symptoms and physical signs of ordinary pulmonary phthisis. I gave him medicine, and told him to see me again in a fortnight; about five or six days

* Delivered before the East Surrey Branch of the British Medical Association at Reigate, October 9th, 1902.

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later his friends came to see me and said that he had suddenly passed a large quantity of blood *per rectum* and died. I asked for and obtained a necropsy. The phthisis was there right enough, of an ordinary type, but the intestines from stomach to anus were full of blood, the exact source of which was at first quite inexplicable, as I could discover no gross lesion in the stomach (its obvious *primary* source); closer scrutiny of the viscus, however, showed me at the cardiac end a hole, about the size of an ordinary pin's head, leading directly into a small vessel; beyond the hole or round it there was no trace of any ulceration visible to the naked eye. At the time I thought I had possibly a peculiarly situated single focus of tubercle to deal with, and allowed myself to say that I had seen a case of tubercular ulcer of the stomach. I removed the piece of the organ with the hole in it, and intended to have sections cut; but unfortunately the specimen got lost, and so the case remained really unexplained till further evidence accumulated of what I now believe to be its real nature.

During the last three or four years in the post-mortem room I have found in at least two or three persons, in whom there was no suspicion of tubercle at all, a precisely similar condition. The history of the last one, by the kind courtesy of Mr. Rigby, I am enabled to give. The body was that of a girl æt. 22, a barmaid. Her previous history is unattainable; but so far as the notes go they are not incompatible with the supposition—they do not specifically state that it was so—that she was one of that very common type of anæmic girls who suffer from indigestion and pain after food; at any rate she was admitted looking very blanched, as a case of gastric ulcer with hæmatemesis, and transferred at once to Mr. Rigby for operation. The history of her present acute illness was as follows:—She had had a bilious attack on Thursday, vomited a pint of blood on Friday, is said to have had several fainting attacks on Saturday and Sunday, and on Tuesday vomited another half-pint of blood, so that there was a reasonable suspicion that she would bleed to death if something radical were not done to check the hæmorrhage. Mr. Rigby operated. The notes run—"No ulcer was found, but a villous-looking patch about the size of a shilling was found, from which the blood seemed to be coming; this patch was sequestrated by a purse-string suture, etc., etc." (From my immediate point of view the surgical procedure is of no particular interest.) At first she did well, but suppurative parotitis supervened with œdema of the glottis, necessitating tracheotomy. She struggled gamely with these difficulties, but on September 24th she vomited blood again, and died on the 25th. At the post-mortem I found the surgical situation perfect so far as the stomach was concerned (except that the sutures were unabsorbed and, indeed, unaltered—a point for surgeons to note). The wounds were healed perfectly, and the old affected region effectually sequestrated; but near the pylorus, some inches from the old spot, was a small pin's-head-sized hole leading directly into an open vessel, with no trace to the naked eye (I think this time I shall succeed in getting some microscopical sections made) of any ulceration or swelling, or other pathological condition than a simple solution of continuity in the mucous membrane. The histories of the other cases differ a little from this one, but the post-mortem appearances were identical in all. Such is my definite evidence of facts from the deadhouse, but in the living the evidence is nearly as strong.

For the last four or five years Mr. Moullin has operated for me upon several cases of gastric ulcer, the operation being demanded for various reasons which we may discuss later—perforation, suppuration, hæmorrhage, life a burden, etc.; but amongst those upon whom he has operated for hæmorrhage it is very noteworthy—and I believe his experience coincides with that of all my surgical colleagues—how frequently he has to report, "I found no ulcer, but only a small hæmorrhagic or petechial-looking spot, in the last case a slit-like appearance, which I was forced to conclude was the origin of the hæmorrhage. I tied that up, and she has done well."

I am therefore, on this evidence, forced to the conclusion that this erosion form of ulcer is of much more frequent occurrence and of much more clinical importance than has been generally admitted, for I believe that it is the condition which is primarily at the bottom of all cases in their early stages; that, in other words, it is the starting-point of most, if not all, ulcers of the stomach of any form whatever.

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PATHOGENESIS.—I have, after a good deal of digestion of the writings and experiments of others, coupled with my own clinical experience, always been inclined to attribute ulceration of the stomach to inflammation and swelling of the small collections of adenoid tissue beneath the mucous membrane, and the above evidence of erosions strengthens my opinion considerably, so that I now accept it *finally* as an hypothesis which has now been proved to be a fact by observation. The collections are well known to the microscopist, but are practically inaccessible to the naked eye, so that if one were slightly enlarged by inflammation and then suppurated, it would leave behind it as the base of the small ulcer just such a hole as I have found; the result of such inflammation would easily be hæmorrhage or a gross ulcer, according to whether the original focus of trouble was close to a fair-sized vessel or not, and according to the severity and extension of the ulcerating process.

DIAGNOSIS.—The next question that arises is, how far does the recognition of the existence of mere erosions influence our opinions when the question of diagnosis arises? It must be in the experience of all of us to have met with cases in which all we have been able to say at first is that dyspepsia is present, and the dyspepsia has later on been followed by severe hæmatemesis, and possibly by all the classical signs and symptoms of ulcer. Can we, in the light of my present text, avoid such incidents? It may be a very lame conclusion to come to, but I fear I am obliged by practical evidence to say that in ordinary life (as we find it with the claims of duty calling every one, no matter what his or her social position may be) such incidents or accidents will, and must, remain inevitable in a certain proportion of cases, for it is simply impossible to send every patient to bed for even two or three days who comes to us complaining of indigestion, and yet that is what the position comes to mean if we would act with absolute safety; for I am quite convinced that patients with these erosions do not complain at first of anything but the mildest symptoms of indigestion. Time after time I find, on looking up my old notes of cases of hæmatemesis—and such or perforation are the ones that a consultant is likely to be called to as urgent cases,—that, even on the most careful inquiry, a little wind or fulness after food, a good preliminary appetite too easily satisfied, slight pain sometimes at once, sometimes delayed for an hour or so after food, are all that has ever been noticed. Such patients are not fit to be very thoroughly tested in regard to localised pain on pressure; but when I make the statement "that when erosions only are present there is no localised tenderness on pressure, only a generalised tenderness of the whole epigastrium, that when a definite gross ulcer has been present there has been this localised tenderness," I am not stepping outside any actual observations noted down, though I am quite willing to admit that it is based more on negatives than on exact positives; a risky position, I admit, but one which can lead to no harm, and may help to the avoidance of accidents in some few cases. The position I take up is this: when we see a patient between, say, fifteen and forty complaining of indigestion without any definite exciting cause, we can neither affirm—without hæmorrhage—nor deny the possibility of erosions being present; and if we remember the possibility, we shall be inclined, whenever it is at all practicable, to send the patient to bed for a few days, and either starve him (or her more frequently) completely, or feed on pulp and slops. If hæmorrhage has occurred in a case with short duration of symptoms, I go so far as to say that erosion is the most likely condition present. We must, however, take the elements of diagnosis and consider the various groupings of them, for it is in the association and linking together of the symptoms, the symptom-complex, rather than on the individual items that diagnosis mainly rests. The items themselves are—

1. Age.
2. Sex.
3. Pain or discomfort.
4. Hæmorrhage.
5. Perforation.
6. Duration: (*a*) over a length of time; (*b*) in an attack.
7. Severity of symptom-complex.
8. Local tenderness.
9. Vomiting, and the nature of the vomited matter.

It would occupy too much of your and my time and patience to follow out every possible combination of these 1, 2, 3, 4, etc., at a time; but I think we may very usefully consider the matter from the opposite end, and take some of the various diseases

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known to occur in the abdomen which require to be distinguished if we would treat our patients on any rational basis.

Movable Kidney.—It may seem rather far-fetched to suggest that a movable kidney may simulate gastric ulcer, but on at least three separate occasions I have had some difficulty in determining how much of the pain after food, and the nausea in youngish women, was due to the movable kidney, which was undoubtedly present, and how much to a pathological condition of the stomach. On one of these cases I had nephrorrhaphy performed, which produced a total and permanent (at least, it is two years since the operation, and she has had no return of symptoms) cure. The patient was a lady whom I had several times ordered to bed and fed upon pulp and slops; each time she improved, but almost at once was as bad as ever again on resuming her duties. One day I was astonished to find a kidney very evidently in a position where it had no business to be, and very easily shifted about. Shortly afterwards the other two cases occurred, and I then began to think that the kidney trouble might have something to do with the symptoms. Accordingly I advised the first patient to submit to the operation; this she did, and has had much cause to congratulate herself on the result. I have lost sight of the other two cases, but they may yet return.

Acute Gastritis.—The special features of this affection are that it practically never occurs (under circumstances that necessitate differentiation from ulcer) in young subjects without a fairly obvious cause in the shape of too much "lobster salad" or some equally indigestible material, or something obviously irritating to the stomach. It is very sudden in its onset, and associated with much nausea and retching; points in which I am led to believe it differs markedly from gross gastric ulcer, though possibly resembling multiple erosions. The appetite is in abeyance from utter distaste for food, whereas in gastric ulcer the appetite may be good, or, if said to be bad, it is because it is frightened away either by the bleeding or by a pain which has been present for some days or weeks, or longer in one spot, not generalised.

A Bilious Attack.—I cannot define this condition, as I am rarely in a position to see one, but in adults my impression is that it is the same thing as acute gastritis, and the "bilious" part of the name has arisen from the sole fact that the vomit becomes almost pure bile, owing to *anti*-peristalsis reaching the duodenum. In young persons I believe a condition does exist which has the same phenomena during its presence as those of acute gastritis in adults, but it is brought on by very slight causes indeed, generally of a reflex—not direct gastric—origin, a sort of neurosis allied in its primordial nature to epilepsy, the distinguishing feature being the history of a "delicate stomach" quite from early childhood or even infancy. The severity of the attacks begins to diminish at the age of, say, fifteen to twenty, when ulcers begin to intrude into the diagnostic horizon. Should an attack of such a nature, *i. e.* without obvious prodromata, occur in an adult, the nervous system should be carefully examined for tabes and other cord diseases, tumours of the brain, meningitis, etc.

Acid Dyspepsia.—Accepting, as I think we must, the existence of such a disease, the following constitute its symptom-complex, viz. the patient is an adult, say from thirty to fifty; pain rarely or never comes on till two or three hours after food, it is much, and that almost at once, relieved by vomiting, and is also relieved very rapidly by the ingestion of some food or plain liquid diluent. It requires to be separated from chronic gastric ulcer, which I think can usually be done by the *combination* of the presence of intense general pain and the absence of a lump to be felt, or local tenderness to be elicited. If it is present without complication, there will, too, be no dilation of the stomach elicited by manipulation without artificial distension. If a chronic ulcer should complicate it, then dilation is likely to be a marked feature.

Positive Diagnosis.—The above are the principal separable affections which require, as far as possible, to be differentiated from gastric ulcer. In speaking of the positive diagnosis of the condition, I have very little to add to the classical descriptions which are given in all text-books; but I think it quite possible that much has to be subtracted from those descriptions, or at least modified, for I have come to believe that in young subjects, say under thirty, without some positive physical sign, such as lump to be felt, *localised* pain and tenderness on pressure, etc., hæmorrhage *per se* and standing alone is more likely to be due to the erosion spoken of above. This point, however, is not worth labouring, because, except for the

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question of surgical interference, the treatment for ulcer and erosion is identical, and erosion is an early ulcer.

I have once had the extreme pleasure—I must use the word, for it is pleasant to be right and to hope that one's *tactus eruditus* has not been cultivated in vain—of proving that I was right in diagnosing that a gastric ulcer had tried to perforate and become adherent to the left rectus muscle in its efforts,—there was a curious feeling of something at the back of the upper part of the muscle preventing its orderly contraction; operation showed that I was absolutely correct, and I think I saved a life, or at least years of suffering. Further points in diagnosis belong to treatment.

PROGNOSIS.—Prognosis is always so bound up with treatment that it can hardly be discussed apart from it; but, although it may tell against my present views, I cannot refrain from giving an outline of two cases which I saw fourteen and thirteen years ago respectively. (1) Miss C—, æt. 25 (in 1888 when seen), had had dyspepsia a fortnight, but had been anæmic for a long time, was attacked with sudden severe pain and localised peritonitis; perforation took place, I think, without a doubt, but she got well without operation, and came to see me in 1900, when she told me that the indigestion had never left her, but so long as she was careful in diet she could enjoy life fairly well. (2) Miss B—, æt. 25 (in 1889 when seen), had suffered for some months from painful dyspepsia relieved by vomiting; on October 18th, 19th, 22nd, and 29th she vomited large quantities of blood. Nothing surgical was done, but she got well, and in December, 1901, I saw her again for another attack of hæmatemesis, and advised her removal to hospital for operation; but when she arrived she refused any interference, and went out as well as before. Between the attack in 1889 and the one in 1901 she had had at least four, and I think she said eight or ten attacks of a similar nature, *i.e.* severe hæmatemesis preceded by indigestion.

One swallow does not make a summer, nor do two such cases as these prove that in either case I was right in my treatment; but they do at any rate suggest to us not to be in too great a hurry to explore the abdomen in gastric ulcer, to exercise a wise discretion in the choice of cases. I think some very interesting and instructive statistics might be obtained by an inquiry answering the following questions:—"Taking all cases in which gastric ulcer is diagnosed, what proportion of them have multiple attacks?" and "How many attacks have they had?"* "What has been the value of life to them between the attacks and subsequent to the last one?" "Has treatment been medical or surgical?" According to Mr. Mayo Robson and Dr. Byrom Bramwell gastric ulcer is an extremely common disease, and as surgical treatment of it is very modern, there should be no difficulty in ascertaining its prognosis unaffected by surgery.

TREATMENT.—To prevent confusion and repetition in the discussion of the treatment of gastric ulcer, we must take it in separate divisions, with artificial limitation better marked in the treatment than in the natural history of the affection.

The first group I make from this point of view is a very large one, viz. those cases in which dyspepsia is the prominent feature, but in which there are no certain indications, such as hæmorrhage, to give a strong lead. It is this group which, as I have already noted, offers such difficulties for diagnosis as to the exact morbid changes present.

Taking the young anæmic girls first, I have one point to urge, viz. that I never yet have found a case which was not improved by iron given at once, provided the tongue was not badly furred; in my experience the tongue in these girls is generally pale and flabby and large, and not often dirty. It is generally stated that the stomach should be cleaned up by bismuth, etc., before iron is given; but I cannot say that this accords with my experience, with the sole proviso regarding the tongue mentioned above, and I am inclined to think that it hardly accords with one's ideas of the best means of treating erosions, which I believe depend more upon some constitutional dyscrasia (in the cases under consideration anæmia) rather than upon any local irritation; much the same as the corresponding affection of the throat, often called a relaxed throat and pharyngitis, as opposed to follicular tonsillitis, depends upon a similar causation. Except for the very great importance

* Mr. Moullin has had this question answered by an appeal to the statistics of the London Hospital for five years, when he found that out of 500 cases diagnosed as gastric ulcer 116 had had one previous attack, 41 had had two, 15 had had three, and 39 had had four.

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of bed, I do not think this group, whether anæmic or not, offers any new or special features in treatment for me to advance; but the rapid subsidence of all trouble directly the patient goes to, and remains in bed is little short of miraculous, and it is this fact which makes me say that whenever it is in the least degree possible young patients with indigestion without a very definite exciting cause, or even with one, should be put to bed. The next question is, When should we let such a patient get up? I believe that the stomach will heal from erosions as rapidly as a throat will heal from congestion, and that in three or four days it is probable that danger is past; but if any doubt still lingers in your mind, there is a very simple plan that I learned from the practice of Dr. Samuel Fenwick, viz. to get the patient to swallow a little salt solution (℥j of salt ad ℥ij of water) in the morning on an empty stomach; if there is still any raw place this will cause a good deal of temporary pain, and will also serve to show whether the condition has passed on to actual gross ulceration or not, and whether, if it has, the ulcer has healed. The pulpy diet, with which you are all as familiar as I am, I pass over with only one remark, but that one I wish to make as emphatic as possible: it is that raw milk should not be given alone; junket, broken-up custard, soft bread and milk, etc., may be given in small quantities at a time, but the patient must not be allowed to drink ordinary raw milk, owing to the large, hard (for the stomach) curds that are produced. To allow milk is a dangerous proceeding, owing to the fact that the public think "milk" means drink as much of it as you like at any time, and they will give it behind the doctor's back.

The second group is a very difficult one to discuss from the point of view of treatment, and I may say at once that I do not think we have yet sufficient experience to dogmatise on it. The group itself is that one in which we feel sure that an acute ulcer is present; and it has three separate types:

(*a*) Ulcer diagnosed by local tenderness, etc.

(*b*) Ulcer diagnosed by hæmorrhage.

(*c*) Ulcer diagnosed by perforation.

Type (*a*), I suppose we shall all agree, must be put to bed and treated, so far as general management is concerned, diet, etc., absolutely on the lines of Group I. One principle here, however, I might emphasise, as I have come to regard it as a vital one, and that is, if vomiting is a prominent feature, we must give the stomach absolute rest—water and medicine are the only possible exceptions,—and starve the patient even for four or five days; in acute cases nutrient enemata may be administered at the pleasure of the medical man, but I think they do very little good. Taking food only excites retching, which is bad for the patient and for the ulcer. When, after starvation, food is resumed, I believe dry food is the best—a dry crust or a biscuit, which cannot be swallowed till thoroughly masticated. I have succeeded admirably in checking the vomiting of bad heart disease by dry food thus given, and I assume it would be good for ulcer. The main thing is to throw to the winds any idea of regular meals or feeding; but let the patients nibble a biscuit or crust whenever it pleases them, but restrict them very rigidly in quantity.

As regards drugs, I have been in the habit of thinking that nitrate of silver has given me as good results as anything else, but doubtless bismuth is very valuable; but I have nothing new or startling to advocate.

If the patients do not get well pretty rapidly, say in fourteen to twenty-one days, they are very liable to drift into my Group III, to be mentioned presently.

Type (*b*).—It is really on the treatment of hæmorrhage from the stomach that opinions will probably differ most. No one, I suppose, would suggest any active surgical procedures in a first attack of hæmatemesis,—it is astounding what a large amount of blood may be lost, and yet the patients almost invariably do well; but when the hæmorrhage is repeated a second, third, or fourth time, and death seems to be almost inevitable if the bleeding vessel be not secured, it is then that we think the surgeon must step in, and, as a matter of fact, I have summoned surgical aid upon several such occasions; the actual numbers I hope Mr. Moullin will give you. On percentage results of these cases compared with the non-operated ones the question must ultimately be decided, but meantime one is inclined to feel great pleasure in one's successes—perhaps a little unfairly so, by allowing them to unduly outweigh the risks, which seem to be very great in letting things alone, and are not to be despised in permitting a laparotomy.

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I doubt if it is possible to lay down any hard and fast rule, so much depends on the exact judgment we form of the condition of the patient at the time; but if I am to formulate a rule, it would run something like the following:—"If a patient has had three serious hæmorrhages within, say, five days to a week, and during that time the pulse has very markedly increased in frequency, we ought to advise surgical measures." I shall let Mr. Moullin dilate on this point more fully.

The direct medical treatment consists, of course, in bed and absolute rest of mind and body, for which purpose I am sure opium is the best drug; it must be pushed freely, say five or six minims every hour, till the patient is well under its influence, and then in doses sufficient to keep him there. Nothing in the shape of food must be given; but water or ice, *at the request of the patient*, may be given in small quantities as frequently as asked for, but not forced on the attention. An intelligent nurse who will follow instructions, and who does not get frightened and anxious, is here worth her weight in gold. I think any of the *direct* astringent and constringent drugs may be useful, but I doubt the wisdom of ergot unless given in very large doses. Extract of supra-renal seems to give good results in some severe cases, as it would appear to have a marked local as well as distant action.

Type (*c*).—That cases do occur in which a limited perforation occurs, and yet in which the patient after refusing operation does perfectly well, we must, I suppose, all admit from experience; but, inasmuch as we cannot as a rule tell whether the extravasation will be limited (*vide* a case above in which I was lucky enough to be able to say that it was then limited) or general, the accident of perforation is so urgent and so likely to prove fatal that I cannot discuss any other treatment than laparotomy by the surgeon, and must frankly and openly throw on to the patient or his friends the onus of refusing it. The possibilities in such cases as refuse operation open up too wide a field of discussion for me to say anything more here.

The third group includes cases in which there is a history of at least a year's duration of what the patient calls severe indigestion, including very possibly hæmatemesis at some time or other, very severe pain in the abdomen, and probably some vomiting. As I have already indicated under diagnosis, it is very important in this group to make up one's mind whether one has to deal with a gross organic lesion of the stomach or with functional trouble only (acid dyspepsia, etc.); but for my present purpose we must assume that the former is the case. What, then, are we to do? The first thing is to ascertain what treatment has already been employed, because the cases may be divided into those that have drifted while under treatment and those that have drifted without seeking professional advice. If, on inquiry, we find that the patient has not been to bed and remained there for at least two weeks, he must be sent there and treated carefully in the same way as in the first group. Further procedures will depend on—

(1) The results obtained from such method of treatment.

(2) What is found:

a. A contracted stomach;

b. A dilated one;

c. A lump.

(3) The inclinations of the patient derived from the amount of social crippling.

(4) Accidents—hæmorrhage, perforation, vomiting.

Of (1) we may say that the result is often excellent, and the patient restored to health by compulsory adoption of a line that has been neglected; but, on the other hand, the results are often very disappointing, and then a consultation must be held between the patient and his friends on the one hand, and medical advice on the other, and in which the position must be squarely faced of the improbability of medical measures being successful. The risks and chances of operation must be carefully explained, and the patient finally allowed to have the casting vote.

If hæmorrhage occurs in this chronic form of ulcer, I think operation is as distinctly called for as it is when perforation has taken place.

If we find a contracted or dilated stomach, it seems to me equally imperative to offer the patient the relief which operation will bring, and which, in the nature of things, cannot be permanently afforded by medical measures. Lavage may relieve for a time, but not for long.

If we cannot find any gross evidence of permanent mischief, and have to trust to the complaints of the patient for our diagnosis, it is then our duty to try and relieve these symptoms by medical

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means first. Lavage is the most important procedure for doing this, as it will frequently give relief for a long time. Of drugs I have obtained the most marked relief from bicarbonate of soda, forty grains in a wineglassful of water, about four hours after a meal. Listerine (a drachm in a tumbler of water on an empty stomach) I have known also help very materially. But it is not much use pouring drugs into a foul stomach filled with fermenting food.

If, however, a chronic ulcer be present, *i. e.* if the case be not one of acid dyspepsia, I believe that any and all medical measures will eventually prove ineffectual, and ultimately the patient must choose between an operation and a condition of constant misery and pain. It is this that renders accurate diagnosis so important.

THE ESSENTIALS OF HISTOLOGY. By E. A. SCHÄFER, I.L.D., F.R.S. Sixth Edition. Longmans, Green, and Co., London.

THIS edition contains considerable enlargements, partly additions to the text and partly new illustrations. The chief additions to the text referred to include descriptions of the structure of the central nervous system. In regard to the doctrine of the anatomical independence of the nerve-cells, the ordinarily received teaching of the present day is that nerve-cells are linked together into long chains of cells by synapses, the physiological path being uninterrupted, although the anatomical path is believed to be interrupted. It has been, however, shown by Apáthy that in Annelids the neurofibrils are, in fact, continuous from cell to cell, and are not interrupted at the synapses. If this is proved true for Vertebrates, the doctrine of the independence of the nerve-cell will require modification. Professor Schäfer points out that though the use of the corpus luteum is not known, certainly it has, however, recently been suggested that it may yield an internal secretion, the effect of which is to produce a fixation of the fertilised ovum in the uterine mucous membrane. The author makes some interesting remarks on the experiments of Bütschli imitating the structural appearance and the so-called spontaneous or amœboid movements of protoplasm. Attention is directed to the fact that similar movements, due to changes in surface tension, are brought about in a simple oil drop, or in a drop of oil-clad albumen, when brought in contact with solution of soap or of any alkali.

THE SURGICAL TREATMENT OF GASTRIC ULCER.*

By C. MANSELL MOULLIN, M.D.Oxon., F.R.C.S.,

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THE first point, gentlemen, in connection with this question which it seems to me I must establish is, that an operation for gastric ulcer is not only feasible, but can be performed with a reasonable degree of safety. For this I shall rely entirely upon my own experience.

Through the kindness of my colleagues, and especially of Dr. F. J. Smith, I have been consulted with a view to operation in eighteen cases of gastric ulcer. (This, of course, does not include the cases of pyloric stenosis, hour-glass contraction, perigastric abscess, and the other complications of this disease which have been transferred to me from time to time, but merely those of gastric ulcer.) Of these eighteen, in five no operation was performed, and they all died. In three of these the immediate cause for consultation was hæmatemesis; but the conclusion at which we arrived in all of them was that they had lost too much blood already to hold out a reasonable prospect of recovery, and that they were beyond even transfusion. At the present time, with the experience I have had since, I think I should make the attempt. The fourth case I regret exceedingly. The patient was a young woman who was sent into hospital with the characteristic symptoms of gastric ulcer. I saw her within a few hours of her admission, but from that time all her symptoms disappeared. She was in hospital for a month, and during that time was only sick once, shortly after admission. After the first day or two she did not suffer from any pain, and at the end of a month she went out, apparently perfectly well, on ordinary diet. About six months later Mr. Cuthbert Dixon, of Mare Street, Hackney, wrote to me saying that she had been suddenly seized

* Delivered before the East Surrey Branch of the British Medical Association at Reigate on October 9th, 1902.

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with all the symptoms of perforation, and taken into another hospital for operation. Whether this was due to the same ulcer as that from which she was suffering when she came under my care is an open question. My impression, for reasons that I will give later, is that it was not; that it was another, which had developed subsequently. But I cannot help thinking that if I had operated on the first occasion, and performed gastro-jejunostomy, the perforation would not have occurred. The fifth case refused operation. I am not quite sure that I am right in speaking of her as dead. She may still be existing, but some months ago, when I made inquiry about her, Mr. Hirsch, of Woolwich, wrote to me that she was reduced to the state of a skeleton, completely bedridden, only kept alive by nutrient enemata, and far too ill and weak to stand anything in the way of operation even if she gave her consent.

In the thirteen others an operation was performed; in nine for hæmatemesis (six of these were in so grave a state that transfusion had to be performed either at the time of the operation or a few hours later); in four for persistent pain and vomiting; and of these thirteen, two died. They were both patients in whom the loss of blood had already been extreme, and I have no hesitation in saying that, had the operation been performed at an earlier date, before so much blood had been lost, they would, in all human probability, have been saved. There is so great a similarity in their history that I will not trouble you with the notes of both; but I feel that I am almost bound to give you an account of one of them, in order that you may understand to what a desperate state they had already been reduced.

The patient was a woman æt. 24. Two years previously she had had a bad attack of hæmatemesis, and she had suffered from indigestion ever since. She was admitted on July 11th, 1902. Six days before that date, and again the day before, she had vomited half a pint of blood. On the 12th, in hospital, she vomited again about the same amount, and, in addition, there was continuous melæna. The patient became very anæmic, yawning, and restless. The pulse-rate ran up to 140, and the next day, as there was no improvement, it was decided to operate. On opening the abdomen and examining the stomach a spot was found on the posterior surface, near the cardiac end of the lesser curvature, where the peritoneum was opaque and thickened, and the sero-muscular coat could not, when pinched up, be separated from the mucous coat beneath. Corresponding to this point, when the stomach was opened there was a well-defined chronic ulcer, with a yellowish base nearly an inch in length and one third of an inch in width, surrounded by softened and swollen margins, which hung over it and partially concealed it. The floor was formed of the muscular layer, and the sides of the superficial ones all matted together. An attempt was made to isolate the portion of the stomach wall on which this ulcer was situated by tying a silk ligature tightly round it from the mucous surface; but the mucous membrane was so softened that the ligatures cut through, and the walls were so thickened that, in spite of several attempts, it could not be done. Accordingly silk sutures were passed from the mucous surface, through and through the tissues at the base of the ulcer, and the contiguous ends of the sutures were tied together so as to effect approximately the same result. Additional sutures were passed on the outside through the sero-muscular coat, and the wounds in the anterior wall of the stomach and the wall of the abdomen were closed in the usual way.

The patient was very much collapsed. She had lost a large amount of blood, chiefly by the bowel, and, owing to the ligature slipping, the operation had taken nearly an hour. Mr. Lett, the Surgical Registrar, transfused two pints of saline fluid into the basilic vein during the operation, and a pint and a half more was poured into the peritoneal cavity. But in spite of this, and of a very large enema (more than a pint) of coffee, beef-tea, and brandy immediately after the operation, Mr. Moore Ede, my house surgeon, to whom I am indebted for these notes, found her so collapsed that evening that he had to transfuse her to the extent of three pints more. After this she rallied, and for the first four days all went well; the vomiting ceased, and the bowels were opened freely. On the morning of the fourth day parotitis set in. The same afternoon the patient was seized with a sudden pain in the right iliac fossa. The next day the vomiting began again. The abdomen remained soft and flaccid, and moved with respiration, and the bowels continued to act; but the patient grew weaker and weaker, the lower part of the abdomen

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became tender on pressure, and it was evident that peritonitis had set in. Reopening the abdomen was contemplated, but the pulse was too weak, and the patient died on the eighth day after the operation. At the post-mortem examination there was general peritonitis, due to the escape of the contents of the stomach through the anterior wound, which showed no sign of healing, the stitches having cut their way out in several places. There was no leakage from the posterior wound at all. The patient had been reduced practically to that condition to which physiologists, when experimenting upon frogs, have given the name of a salt frog, one, that is to say, in which every particle of blood has been replaced by salt solution,—a condition most valuable, doubtless, from the point of view of pure physiology, but not one calculated to facilitate the repair of grave abdominal injuries. Had the operation been performed earlier, before this stage had been reached, I see no reason for doubting that the result would have been entirely different.

In the other fatal case the conditions were almost the same. There was an artery gaping widely on the floor of a chronic ulcer. The patient had already lost an enormous amount of blood. Transfusion was performed, and for a few days all went well. Then peritonitis set in, and on the eighth day the patient died. At the autopsy the wound in the stomach wall was gaping widely, and showed no evidence of repair. In this patient, however, it was noted, in addition, that the kidneys were in a state of advanced interstitial nephritis.

Had these two cases been left to themselves, I do not think there can be a doubt that they would have died, probably before the eighth day. I do not like to assert the same of the seven other cases of hæmatemesis in which I have operated, and which survived; but I think there can be little doubt about it in the case at least of those four in which it was judged necessary to perform transfusion at the time of the operation or immediately after. Their condition was undoubtedly extremely critical, and there was no evidence that the hæmorrhage would not continue.

I need not give the steps of the operation in detail, but there are a few points of some importance to which I must allude. The stomach is not washed out beforehand. If, when the abdomen is opened, it feels as if it were full, the contents can be pressed out through the pylorus, or sponged out when the wall has been incised. I usually open the abdomen in the middle line because it is impossible to say at which end of the stomach the ulcer may be situated. When this is known (occasionally the presence of a swelling at the pylorus gives a clue) I prefer a lateral incision, separating, without cutting, the fibres of the rectus. The first thing is to draw out the stomach, and inspect the anterior surface. If there is nothing abnormal to be seen, an opening is made in the transverse mesocolon, at some spot where there are no vessels, and the posterior surface of the stomach drawn forwards and carefully examined in the same way. There may be adhesions or a scar, or there may be merely an opaque spot on the serous surface, indicating that inflammation has been going on in the immediate neighbourhood. In two instances in which I found this, I was able to prove that the walls of the stomach were matted together. On pinching up the sero-muscular coat the mucous coat did not slip out from between the fingers as it normally does. Sometimes by doing this it is possible to obtain a clue to the position of the ulcer before opening the stomach itself.

If nothing abnormal can be found, the stomach should be drawn well forward into the wound, packed round with two large flat sponges, one above and one below, and an opening made into it, so that the interior can be examined. In most instances there is no need to introduce the sponges into the peritoneal cavity. They may be laid flat upon the abdominal wall and clamped together with pressure forceps, so as to embrace the protruding portion of the stomach. The wall of the stomach is then incised, usually in a direction parallel to the vessels, as this gives sufficient room and does not cause much bleeding. It might be thought that if gastro-jejunostomy were found necessary an incision in this direction would not lie in the same line as the bowel. But the long axis of the stomach runs much nearer to the vertical line than is usually represented, and, as a matter of fact, I have never found it occasion the least inconvenience. The edges of the incision in the wall of the stomach are then held apart by four pairs of pressure forceps (which also control the hæmorrhage, if there is any), and the interior thoroughly and systematically reviewed. That portion of the posterior wall which lies opposite the

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opening is, of course, seen with ease; and those portions of the posterior wall which lie near can easily be brought into view. For the rest, it is best to make use of a large vaginal speculum, and by introducing the fingers of one hand into the lesser peritoneal cavity below the stomach, to manipulate the posterior wall in such a way as to make it pass across the end of the speculum bit by bit. By doing this and by moving the speculum, the whole of the interior of the stomach can be inspected without difficulty, with the exception of the cardiac orifice. This can only be seen in thin patients with a very soft abdomen.

An acute ulcer is usually described as circular in outline with sharply-defined edges, and in general shape resembling that of a truncated cone. This statement is true of an ulcer as seen in the post-mortem room; but during life, judging from the cases I have had the opportunity of examining, it requires a good deal of modification. In one case I found a raw granular surface, slightly depressed below the level of the rest, and bleeding freely. It looked as if the upper layers of the mucous membrane had been scraped away. But in most, all that could be found was what appeared to be a linear or stellate fissure with soft and swollen edges, from between which blood kept oozing in a steady stream. The discrepancy is simply the result of the vascularity of the walls of the stomach. During life, and especially while digestion is going on, the appearance of the mucous membrane of the stomach is very different from what it is after death. When there is an acute ulcer, owing to the inflammation that surrounds it, the difference is greater still. Immediately around the ulcer the mucous membrane is swollen, congested, and raised above the level of the rest. Its colour is bright red, though the interior of the stomach may be paler than natural, owing to the blood that has been lost. The sides of the ulcer are thickened and œdematous, and so soft and swollen that if the ulcerated surface is small, they meet together over it, and cover it in until it appears to be nothing more than a fissure. After death, when the blood-pressure has fallen, especially in a case in which the vessels have already been emptied by previous hæmorrhage, all this thickening disappears, and the ulcer assumes the crater-like form which is usually described.

Chronic ulcers present a greater variety of appearance. There may be little more than a deep fissure at the bottom of one of the rugæ, or there may be a flat surface, nearly as large as the palm of the hand, composed of the muscular and serous coats matted together, or of the liver or pancreas, or some other of the neighbouring structures tied down by adhesions. In one case I found an open vessel on the floor. The edges of the ulcer may be steeply cut, or shelving, or raised above the level of the rest, but there is seldom such an amount of swelling around as when the ulcer is acute, because, owing to the organisation that has taken place, all the coats of the stomach are bound down by dense adhesions. Sometimes, however, the congestion and œdema assume considerable proportions. In one of my cases a ligature could not be applied, owing to this cause; and another has been recorded in which, for the same reason, inversion and suturing of the ulcer could not be carried out. A few days later, at the post-mortem examination, no trace of this swelling could be found. It was caused entirely by congestion and inflammatory œdema.

The mode in which the ulcer should be treated is a most important matter. Wherever it is possible it should be dealt with directly. Gastro-jejunostomy should be reserved for cases in which this is impossible; those in which, owing to the repeated recurrence of gastric ulcers, it is suspected that there is some peculiar condition of the stomach that gives rise to them, and those in which the seat of hæmorrhage cannot be discovered. Gastro-jejunostomy will not stop the bleeding from a branch of the gastric arteries, which is the usual source of the hæmorrhage; and, as a matter of fact, there are at least two cases on record in which it was performed with this idea and failed.

No one method, of course, will answer. In two cases, in which I found merely a bleeding fissure such as I have described, I successfully strangulated the fissure and the mucous folds on each side of it by passing a silk suture beneath and around it, and tying it tightly. In three cases, in which the ulcer did not involve the submucous layer, I excised the portion of the mucous membrane involved, and drew the edges of the wound together with sutures. In one case I excised the affected portion of the stomach wall, removing the whole thickness. In two others I tied a ligature

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tightly around the ulcerated portion of the stomach from the mucous surface, pushing the wall of the stomach inwards with my finger, so as to make it project into the interior in the shape of a cone. A few Lembert sutures were passed through the sero-muscular coat afterwards as a safeguard. Both of these cases were completely successful. And in one, as already described, in which I could not carry this out, owing to the thickening and softening of the wall, I sequestrated the affected portion by ligatures passed through and through, tying the contiguous ends together. The wound in the anterior wall of the stomach and that in the abdominal wall are closed in the usual way. The whole operation seldom takes more than three quarters of an hour. The greatest care, of course, is taken to maintain the patient's temperature. Strychnia and caffeine are given hypodermically before the operation, and as often afterwards as may be judged advisable from the condition of the pulse. Transfusion (intra-venous) was required on six occasions. In one I left a pint and a half of saline fluid in the peritoneal cavity, and in another I injected some hot brandy and water through the pylorus into the duodenum. Immediately after the operation an enema of at least a pint of beef-tea, brandy, and black coffee is given, and as a general rule, nutrient enemata are kept up for two days. As soon as the vomiting due to the anæsthetic begins to subside, small quantities of hot water are given by the mouth; and if this is well borne, peptonised milk is given instead. By the next day, as a rule, the patient is able to take four ounces at frequent intervals without inconvenience. The bowels are always made to act at the end of forty-eight hours, though there is sometimes considerable difficulty, one patient, in whom an ulcer had been excised for persistent pain, required no less than sixteen grains of calomel, two ounces of castor oil, and two large injections containing turpentine before there was any result.

So much, gentlemen, for the question of the possibility of operating upon cases of gastric ulcer. The next question is whether such a proceeding is advisable, and if so under what circumstances.

I may at once disarm criticism by saying that I have not the slightest intention of advocating operation as a general mode of treatment for ulcer of the stomach. Ulcer of the stomach is, and always will be, more medical than surgical. And I admit freely and fully that the cases of gastric ulcer that are admitted into hospital, and into the London Hospital in particular, are much more severe, taking them all together, than those that occur in private practice. The proportion of deaths in private is much lower than it is in hospital, and very much lower than that of those cases for which a surgeon is called in. But all this has nothing to do with the question I am considering now. So far as my present subject is concerned, I have only to deal with the gravest cases, such as are admitted into hospitals, and only indeed with a small proportion of these. No question of operation could ever arise in connection with a case of gastric ulcer, the symptoms of which were not sufficiently severe to cause its instant admission as an in-patient. These cases, therefore, do not enter in any way into the argument. And further, a very large proportion of cases that are admitted into hospital must also be ruled out. They may present symptoms sufficiently severe to require treatment as in-patients, but their symptoms are never grave enough for the question of operation to be so much as raised. The cases that are left, the gravest of the grave, form probably but a small proportion of the whole; but they are the only ones that are of the least value in instituting a comparison. If these, the really grave cases, are taken by themselves, separately from all the rest, and an estimate made of the rate of mortality among them, it will not be found to compare at all favourably with the rate of mortality of the same class of case after operation.

Dr. C. V. Bulstrode has very kindly collected for me the statistics of all the cases of gastric ulcer admitted into the London Hospital from 1897 down to August, 1902. They were just 500 in number, 98 men and 402 women. Out of this number no less than 48 (nearly 10 per cent.) died from perforation and peritonitis; 13 (or 2·5 per cent.) from hæmatemesis; and 28 (5·5 per cent.) from other causes. The total number of deaths was 89, or approximately 18 per cent., taking all the cases together. Had all the slighter ones been left out, and only those reckoned in which the gravity of the symptoms suggested the possibility of operation being advisable, the rate of mortality, bad as it is, would have been far higher.

The question of the actual rate of mortality,

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however, is not the only one that requires to be considered. Eighty-two per cent. of the cases of gastric ulcer admitted into the London Hospital were discharged. It would be exceedingly interesting to ascertain what proportion of these were cured; that is to say, left the hospital perfectly well, without suffering pain or inconvenience when taking ordinary diet, and remained well. Such a thing unfortunately is not possible. Some idea, however, can be formed by ascertaining the number of patients admitted into hospital with gastric ulcers who had already been treated for and cured of similar attacks. Dr. Bulstrode has done this for me, and has calculated that out of the 500 cases no less than 214 (42 per cent.) had suffered in this way previously. In 116 there had only been one previous attack, in 41 there had been two, in 15 three, and in 39 four or more. In other words, gastric ulcer recurs or relapses in at least two fifths of the cases which are apparently cured.

Even this is not all. These figures do not include any of the cases admitted suffering from what may be called the late sequelæ and complications of gastric ulcer. They do not take into account the cases admitted for pyloric stenosis (which Gerhardt and Warren both estimate at 10 per cent. at least), hour-glass contraction, gastric dilatation, chronic indigestion due to the presence of old adhesions, or those numerous other disorders which are caused by the cicatrisation of ulcers healed long since. Nor do they take into account those cases in which carcinoma has developed at the site of an old ulcer. Hauser estimates the proportion in which this occurs as at least 6 per cent.; and though others do not put it quite so high, there can be no question that it is sufficiently serious to merit consideration.

When allowance has been made for all these after-troubles, for the 18 per cent. of deaths and the 42 per cent. of relapses, it must be admitted that the proportion of cases of gastric ulcer, sufficiently severe to be admitted into hospital, which are cured, in the full sense of the term, is not a very high one; not nearly so high as is generally imagined. What would it be if all the slighter cases that get well so rapidly were left out, and only the very grave ones taken into consideration? Yet these, the very grave ones, are the only ones that are taken into consideration when the question of the result of operation in cases of gastric ulcer is raised.

So much, gentlemen, for the results of the present method of treating the graver cases of gastric ulcer. I do not think I am going beyond the mark when I say that there is ample room for improvement. If only all these graver cases, or certainly the gravest of them, were treated by operation, not only would many more lives be saved, but a vast amount of suffering would be avoided. There remains the question of the indications for operation.

So far as acute ulcers are concerned, these are either threatened perforation or hæmorrhage.

Our knowledge of the premonitory signs of perforation is very small. As a matter of fact, there may be none at all. There are many instances on record in which a patient who apparently was suffering merely from slight dyspepsia has been suddenly seized with agonising pain in the epigastrium, due to the rupture of an ulcer, the existence of which had not even been suspected. Nor is this surprising, since it is now established that the greater portion of the wall of the stomach is as insensitive to pain and as devoid of ordinary sensory nerves as the wall of the bowel. This, it is well known, can be cut with scissors, or pinched up with artery forceps, or cauterised, without the patient even being conscious of it; and though opportunities for demonstrating it are not so common, the same thing is true of the greater part of the wall of the stomach. A local patch of necrosis, suddenly developed, may involve the whole thickness of the wall, so that it gives way with the least strain, without having occasioned previously a moment's inconvenience. The severity of the pain that comes on after food, and the time when it begins, are of little or no value as an indication. The former is very difficult to estimate, especially in the class of patient in whom gastric ulcer is most common, and may be nearly as great in simple dyspepsia. The latter depends upon the portion of the stomach involved, the activity of its movements, and the rapidity with which the neighbouring sensitive portion of the parietal peritoneum becomes affected by irritation spreading from the ulcer. It will, for example, occur earlier when the ulcer is at the cardiac end, owing to the proximity of the diaphragm and the readiness with which the peritoneum on its under

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surface is affected through the short gastro-phrenic omentum, than when it is at the pyloric end. I am disposed, however, to attach much importance to local tenderness on pressure, especially when it is associated with cutaneous hyperæsthesia in Head's epigastric triangle. This is an indication that there is sufficient irritation spreading from the surface of the sore to cause persistent tenderness of the neighbouring portion of parietal peritoneum—a tenderness which is reflected down the corresponding cutaneous branches of the spinal nerves. When this is present and well marked, it may be taken as evidence not of the existence of an ulcer, but of the irritation of the ulcer, and that the sore is not only not healing, but in all probability is spreading, and is therefore dangerous from the point of view of possible perforation.

Hæmorrhage is present in the majority of cases of acute gastric ulcer, and is only to be taken as an indication for operation when it is either excessive or long continued. In estimating the amount of blood that is lost, it must be remembered that that which passes away by the bowel must be counted as well as that which is vomited. In one of my cases the patient only vomited blood once, but the melæna was so frequent and profuse that she was on the verge of death from loss of blood at the time of the operation.

Hæmorrhage in the case of a gastric ulcer may be capillary, venous, or arterial. In the vast majority of instances there is no means of telling one from the other. From whatever source it comes, the blood collects in the stomach, and when the amount is sufficient to excite peristalsis it is either vomited up or passed on into the bowel. Putting aside cirrhosis and carcinoma, it may be said with a fair degree of certainty that when hæmorrhage from the stomach is serious the blood nearly always comes from a branch of one of the main arteries. Fatal capillary hæmorrhage has been described many times, it is true, but this cannot be accepted as proved in any case in which it is not shown by injection or other means that the walls of all the larger vessels are intact. The blood nearly always comes from a minute opening in the side of an artery, but, owing to the swelling of the soft tissues which surround the ulcer, it can only find its way out slowly. It has to separate and force its way between the sides of the cleft at the bottom of which the artery lies. This deadens the pulsation, so that when the stomach is opened under an anæsthetic, the blood appears, as I have described already, to trickle out in a thin continuous stream. When food is taken, all the gastric vessels dilate, the pressure rises, and the bleeding becomes so much more free that very serious hæmorrhage may take place from what appears to be quite an insignificant little vessel, such a one as would be easily overlooked unless special measures were taken to find it. The conditions during life, and especially during digestion, are very different from what they are after death. When the blood-pressure has fallen to zero, and the elastic coats of the vessels have contracted to their utmost limit, an artery, even when it is of some size, shrinks up and becomes so small that it is scarcely noticed, unless very carefully looked for, and any opening in its wall is easily passed over.

Hæmorrhage into the stomach is an indication for operation when it is either excessive or long continued. No patient should be allowed to bleed to death if there is a possibility of stopping the hæmorrhage by exposing the bleeding point.

One single hæmorrhage may prove fatal. Fortunately this is very rare in the case of an acute ulcer. Nearly always, if the stomach is placed at perfect rest, the bleeding stops in time if it is the first attack. Not even water, however, may be allowed, and certainly not ice. But though it is the first attack, if the pulse-rate continues to grow quicker and quicker, and particularly if the patient becomes restless, throwing the arms up over the head, and if the respiration becomes interspersed with deep sighs, there should be no further delay, even though there is no vomiting. The hæmorrhage in all probability is going on still, and the blood collecting in the bowel or the stomach. The end in such cases is sometimes very sudden.

If the first hæmorrhage is only moderate in amount, so that the patient rallies well, but is followed by another attack within a few days, in spite of absolute rest, the bleeding point should be exposed and dealt with on ordinary surgical principles without delay. Some vessel is bleeding. The chances are that it is an artery, and that there is a hole in one side of it, a kind of opening that notoriously does not close. The bleeding has stopped once. The clot that plugged the opening has been removed by the action of the gastric

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juice, or the irritation which keeps the ulcer open and prevents its healing has caused a further destruction of the wall, and the hæmorrhage has returned. It is not safe to run the risk of allowing this to happen a third time. Provided the circumstances are suitable and adequate assistance is at hand, the danger of operating is the lesser of the two. Under these conditions laparotomy at the present day is a perfectly safe proceeding. Opening the stomach, if it is done with proper precaution, does not add materially to the risk. There remains the difficulty of finding the bleeding point and of dealing with it. Neither of these is insuperable. The former, I admit, after death often presents difficulties; but it rarely does so during life. The latter, if the ulcer is recent, needs no further mention. It is only in the case of chronic ulcers that have been allowed to persist for years that any real difficulty occurs.

The same argument holds good for those cases in which hæmatemesis returns after long intervals of apparently perfect health. This is the condition which I have described elsewhere as recurrent ulceration of the stomach, and which is often mistaken for the chronic form. Whatever may be its cause, acute ulcers form again and again, each with the risk of hæmatemesis and perforation. That patient whose case I described to you at the beginning of this address, who, six months after leaving the hospital apparently cured, was seized with all the signs of acute perforation, is an instance in point. There may be an interval of many months. Then suddenly a large amount of blood is brought up, or the wall of the stomach gives way, almost without warning. Until the cause that leads to this successive development of ulcers has been ascertained, it is better that gastro-jejunostomy should be performed, and the stomach allowed to recover under the rest this gives it, than that the patient should be exposed to perpetual danger, and live in a state of constant terror that at any minute, without warning, a huge and perhaps fatal quantity of blood may be brought up.

Other indications of scarcely less importance are repeated hæmorrhages, even though they are slight in amount, and constant pain and vomiting. The one slowly but steadily undermines the patient's strength until at last phthisis or some other disease sets in; the other renders life one long misery, and reduces the patient to the condition of a hopeless invalid.

Chronic ulcer of the stomach stands on a somewhat different footing. The difficulty here in the diagnosis is to make certain that there is a chronic ulcer, and that it is not merely a case of severe dyspepsia. If the diagnosis is made, I believe the stomach should in all such cases be explored with a view to ligature or excision of the ulcer, or, failing that, gastro-jejunostomy. Palliative treatment, in the hope that the ulcer will heal of itself without leaving behind it any disabling cicatrix, may be given one good trial if it has not been tried already. But the fact that the ulcer has become chronic is proof that the previous treatment has failed; and if it has failed at the beginning of the ulceration, it is not likely to succeed when the ulcer is thoroughly well established. The longer a chronic ulcer lasts the more the local conditions which attend it, such as the induration about its base and the adhesions to surrounding organs, prevent its healing.

Hæmorrhage, pain, and vomiting, again, are the main indications; and here one single severe hæmorrhage is enough, for in chronic ulcer of the stomach the blood usually comes from an artery of considerable size, the sides of which are held apart by the dense fibrous tissue that surrounds it. But even if these are not absolutely urgent, the persistence of a chronic ulcer is sufficient justification. No patient is safe with an ulcer in the stomach that either will not or cannot heal. Putting aside all the complications that may occur, all of them serious and some of them fatal, there comes a time in these cases when a complete operation is no longer possible, and even palliative ones, such as pyloroplasty and gastro-jejunostomy, very difficult. Every museum is full of specimens of chronic gastric ulcer in which a complete operation would have been impossible, and a palliative one very dangerous, at the time of the patient's death. There was, however, in each a time when a cure would have been comparatively easy. Besides, there is the risk of cancer, which undoubtedly results in a certain proportion of these cases; and finally, there is the constant risk of hæmorrhage and perforation. Some time ago Dr. Wall collected for me the statistics of the cases of gastric ulcer admitted into the London Hospital during five years. Only those suffering from hæmatemesis

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were counted; and, as far as possible, all those in whom the hæmorrhage arose from cirrhosis, malignant disease, and other causes than ulcer of the stomach were excluded. Of those over thirty years of age, no less than 6 per cent. of the women and 12½ per cent. of the men died from this cause alone. If to this is added the number of deaths from perforation, exhaustion, repeated hæmorrhages, subphrenic and perigastric abscesses, and the other complications that occur in connection with gastric ulcer, it must be admitted that the total death-rate is a very serious one, far higher than would be the case after timely operation.

ANÆSTHETICS: A PRACTICAL HANDBOOK. By J. BLUMFELD, M.D.Cantab., Assistant Anæsthetist to St. George's Hospital; Anæsthetist to the Grosvenor Hospital for Women and Children. Medical Monograph Series, Baillière, Tindall, and Cox. Pp. 109, crown 8vo. Price 2*s.* 6*d.* net.

DR. BLUMFIELD has written this small book for students preparing for those final examinations before which a course of instruction in anæsthetics is now rightly made compulsory. The teaching throughout is sound, being practically that of Dr. Hewitt; indeed, the author, in his preface, acknowledges the free use made of the latter's writings. The nature and action of the common anæsthetics are first considered; they are then taken in turn, and the mode of administration of each, and their chief uses are described, and afterwards their various combinations and successions. The dangers and troubles likely to be met with, the choice of the anæsthetic, and the preparation of the patient, are discussed at some length; while short chapters are given on the question of the position of the patient and the administration of anæsthetics to children. An efficient index completes the book. The limited size of the volume prevents a very full description of some of the methods mentioned; in fact, the account of the administration of gas and oxygen would hardly be intelligible to any one unacquainted with its use.

Dr. Blumfeld prefers a C.E. mixture consisting of two parts of chloroform freshly mixed with three parts of ether to the ordinary A.C.E. mixture.

The few slips noticed in reading the book are not very important; but it is to be hoped that the term "chloroformisation" will not come into general use.

PHARMACOPŒIA OF THE PADDINGTON GREEN CHILDREN'S HOSPITAL. London, 1902 (Adlard and Son). Price 2*s.*

THIS little work consists of two parts, first the medicines which are in common use at the hospital for internal and external administration, and secondly a series of directions on the rearing of infants, the treatment of rickets being specially considered. The tendency of the times is shown in the limited number of prescriptions employed and the absence of polypharmacy in those prescriptions. Thus the use of iron is limited to four formulæ, namely, *M. Ferri Acida*, *M. Ferri Alkalina*, *M. Ferri Aperiens*, and *M. Ferri et Potassii Chloratis*, and we are inclined to agree that with these the physician can treat all cases with a suitable preparation of iron. The only indications of polypharmacy present are in the cough medicines, the ingredients of which are sometimes four or five in number, and are rather antagonistic in their action. A large variety of lotions is given in strengths suitable for infants, but we may point out that a *Lotio Evaporans* (p. 24) consisting only of ammonium chloride and distilled water will take a long time to evaporate. A sufficient number of prescriptions for the treatment of diseases affecting the skin, the eyes, the ear, nose, and throat is also included. With this little volume at hand the practitioner will be enabled to prescribe for all the diseases of infancy and childhood, and, by increasing the dose, to treat those of adult life also. The directions for the rearing of infants are simple, and plain, and full. We heartily endorse the statement that no "infants' food" should be given unless by medical order until the child is over the age of nine months. Apparently the medical authorities at the Paddington Green Children's Hospital do not think them necessary after that age in the case of healthy children, for the foods recommended are all such as can be prepared freshly at home. We think that probably the same advice is given at all the London children's hospitals, where the staffs are more engaged in combating the evil effects of proprietary foods than in ordering them. In connection with rickets full directions are given as to the diet, the clothing, and the indoor and out-of-door life of the little patients.

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** Specially reported for the Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE

ON

INFANTILE CEREBRAL PALSIES.

Delivered at the National Hospital for the Paralysed and Epileptic, Queen Square, London, June 24th, 1902,

By HOWARD H. TOOTH, C.M.G., M.D., F.R.C.P.,

Physician to the Hospital.

GENTLEMEN,—To-day I wish to say something about a large and important group of nervous diseases, namely, the cerebral palsies of infancy. At the outset I need scarcely do more than remind you that my subject does not include infantile paralysis, commonly so called, which is a disease of the grey matter of the cord, but that I am speaking to-day entirely of paralytic symptoms which make their appearance secondary to demonstrable disease of the brain, without spinal symptoms, except such as are referable to secondary changes in the spinal cord.

We may divide the cerebral palsies of infancy into two main groups; those which are due to traumatic lesions, and those which are due to disease other than traumatic.

Paralysis of traumatic origin. — Traumatic palsies, or birth palsies, constitute a group of considerable size and diversity of symptoms. The early history of traumatic cases is nearly always obscure, and the first appearance of symptoms much less definite than would be expected.

The lesions of birth palsies are due, as a rule, to some defect in the mechanism of labour. Such defects are generally referable to malformation or undue rigidity of the parts in the mother, giving rise to prolonged difficult labour; but a violent precipitate labour may produce the same effect on the child's brain. The use of instruments has been credited with causing birth palsies, but it must not be forgotten that instruments are employed to facilitate delivery in the very cases

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which, if left to nature, might result in brain lesions; and that though no doubt the unskilful use of forceps may lead to damage of the brain, yet it is not unreasonable to suppose that many a child escapes a birth palsy by the timely use of instruments.

There is an interesting point in connection with the first appearance of symptoms of paralysis. One might think that a brain lesion sustained at the time of birth would present symptoms of paralysis very soon, if not immediately after birth. This apparently is not by any means always the case; if the histories of such cases are to be believed, a considerable lesion of the brain may be sustained at the time of birth without producing symptoms until after the lapse of some weeks or even months. In explanation of this it is probable that the earliest movements of childhood after birth, and certainly those before birth, are not cerebral or purposive movements, but of a reflex nature, and therefore referable to the grey matter of the spinal cord. This seems to fit in with the anatomical fact that the lateral columns of the spinal cord are not fully developed or myelinated at this period. It appears essential for the orderly transmission of voluntary impulses that myelination of the axis-cylinder should be complete, and this is not so until towards the end of the first year.

Another point seems to fit in with this view, namely, that the plantar reflex in children at this age normally presents the extensor response; and the flexor movements, on tickling the sole of the foot, do not make their appearance until the child is about a year old. These facts may explain the late appearance of paralytic symptoms in cases of serious cerebral lesion sustained at birth. The varieties of birth palsies are very numerous, and they may be classified in precisely the same manner as the cases of non-traumatic origin considered in the next section.

Paralysis of other than traumatic origin.—Now to discuss disease of the brain other than traumatic as evidenced by the symptom paralysis. The pathology of these cases will be considered later, but for the present purpose we may state that these palsies are practically always due to disease of the cortex, either of inflammatory or thrombotic vascular origin, or of the nature of an acute cellular degeneration.

1. *Paralyses of inflammatory or vascular origin.*—In this category are included the large and varied group of infantile hemiplegia, monoplegia, and bilateral hemiplegia, diplegia, or so-called spastic paraplegia, together with the associated conditions of epilepsy and degrees of idiocy.

The age at onset may be any age from shortly after birth to two or three years, or even to thirteen years. Two cases described by Sir Dyce Duckworth and myself in the 'St. Bartholomew's Hospital Reports,' 1892, were probably of this nature. The onset of the disease is usually acute, and there is frequently, though not invariably, a distinct illness, so that the symptoms can often be referred to a definite period of onset. That is not always the case, however, and in some cases it is extremely difficult to say when first the symptom of paralysis made its appearance, especially if it comes on very soon after birth, for the reasons I have stated. But there is generally one symptom which marks the onset, namely, convulsions. It is not unusual for such patients to have an epileptic fit, or, what is equally common, a series of epileptic fits, the condition of *status epilepticus* lasting, perhaps, for two or three days, or even a fortnight or three weeks. From the first there may be fever, often with considerable rise of temperature. The early diagnosis of the disease is undoubtedly obscure until the paralytic symptoms appear, and by that time irremediable cerebral damage may have been sustained.

The cerebral palsies may be grouped in three main classes according to the nervous symptoms. The commonest of all, perhaps, is that form in which the lesion is one-sided, infantile hemiplegia. When the comatose and convulsive stage is over, the child is found to be paralysed on one side, the picture differing in no way from that of hemiplegia in the adult. But as the child grows certain distinctive features appear. The loss of power is rarely absolute ultimately; in fact, there is a tendency to regression of the pathological processes in the cortex. The grasp on the paralysed side some years after may be as strong as on the sound side, and yet the child is an obvious hemiplegic. The fact is the orderly performance of voluntary movements may be profoundly interfered with without actual loss of power; and one factor in the production of these disorderly movements is the appearance of varying degrees of rigidity. But this rigidity differs

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from the rigidity of adult hemiplegia in that there is a variable amount of mobility about it, so that the term "mobile spasm" has been applied to it by Sir William Gowers. There are a considerable number of forms in which this may show itself. Where this mobile spasm is present in its extremest form the picture is one which receives the name of athetosis,—constant, almost rhythmical movements of the paralysed limb, aggravated by all attempts at movement. Sometimes the movements have a jerking, purposeless character like chorea, and the condition then has received the name post-hemiplegic hemichorea.

There is another curious form, called "chorea spastica," which resembles rather closely the tremor of disseminate sclerosis, the so-called "intention tremor," which does not come out until the child attempts to perform some definite act.

As in adult hemiplegia, there is a tendency for the leg to recover more completely than the arm; and in some cases the recovery of the leg is so perfect, and that of the arm so slight, that the condition may be almost termed monoplegia. Usually, however, there is some defect of movement in the leg, spastic or athetoid, commonly only a clumsiness, causing the child to trip frequently, and to bring the foot down in walking with unnecessary force. In severe cases the nutrition of the paralysed limbs is profoundly interfered with, so that development is almost arrested, and the limb is much smaller than its fellow. All the tissues of the limb are affected equally, not the muscles only; the skin, for instance, is thinner, and the bones smaller than natural.

I now come to speak of an interesting and fairly common form of distribution of the disease with regard to symptoms, namely, double hemiplegia,—that is to say, hemiplegia on both sides of the body, so that the resulting picture is one of paraplegia. A number of names have been given to this. It is spoken of as diplegia, as bilateral hemiplegia, as double hemiplegia, infantile spastic paraplegia, and Little's disease. This bilateral arrangement illustrates the tendency to a symmetrical disposition of the lesions in the brain. The appearance of spastic diplegia is striking. There is little voluntary power, but it is the spasm which gives the characteristic features to the condition. The rigidity is generally extensor, but not invariably. The arms may be extended straight at the elbow, and the wrists flexed, the fingers in the position known as the "claw-hand." The knees are powerfully extended, spasm of the adductors leads to crossing of the legs, the ankles dorsi-extended, and the toes over-extended, causing sometimes dorsal dislocation of the great toes. The reflexes may be increased, and there may be ankle-clonus, but where the rigidity is extreme the jerks may be difficult to elicit. The plantar reflexes are increased, and the extensor response is generally to be obtained. Sensation is rarely if ever found to be affected in the later years, and in the early stages the demonstration of anæsthesia is scarcely possible.

Here I show you a little girl. There is no history of the origin of the disease, because she is a foundling. She is a bright, intelligent-looking little girl. Notice the condition of her arm; you see it exhibits in a mild degree the mobile spasm I have spoken of. The arm at first appears to be paralysed, but if we ask her to take hold of something she can do it. But notice the grotesque movements she makes in order to take hold of this stethoscope. And when she tries to give it me back she cannot relinquish her hold upon it. That is a typical specimen of a mild degree of mobile spasm, and you can construct an ascending scale of severity from this to the most pronounced form, of which I have not an instance to show you, in which all day long the affected limb is undergoing rhythmical movement; so distressing and annoying are these movements that in one case at least at this hospital the motor area has been removed so as to produce complete paralysis as a preferable condition, with the full knowledge and concurrence of the patient.

These athetosis movements rarely result from lesions of the brain in the adult, while they are so common in children as to be almost the rule. They probably indicate an incomplete destruction of motor cortex, or an attempt at spontaneous cure, by which islands of natural grey matter remain among the diseased tissue. The result is diminished motor power, disorderly voluntary movement, still further impaired by rigidity. Her gait is simply tottering. As the child grows up she will become more and more clever in walking, until at length she may walk well. But the arm will probably never recover. In all probability she was originally a case of infantile hemiplegia.

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The next patient is an instance of infantile spastic diplegia, and one in which there is a history of instrumental labour, but of course it is impossible at this distance of time to say whether the trouble was due to the use of instruments at all. He is now eighteen years of age. From the history given by the mother it appears that the defect was not noticed until the child began to try to walk, at the age of eighteen months. He could not walk until he was three years old. He has had three fits. You will notice that he talks with effort. I think there is the same difficulty in some of these cases in the production of the sounds of speech as there is in the production of the movements of limbs; there is what we may call a certain rigidity of the muscles of speech. One gets all sorts of curious speech effects, from complete dumbness which may never recover, to a simple staccato explosive utterance. There is never true aphasia in these cases. There is a tendency to dribble. He protrudes his tongue with effort. There are constant fine tremulous movements of the head, but no nystagmus. In the upper extremity all movements are performed with effort, and are tremulous. There is more difficulty with the left hand than with the right. The grasp is quite strong, there is no true paralysis. What remains is a difficulty in performing the final movements. He writes quite a legible letter, but there is a tremor perceptible in all the strokes. In the lower extremities there is no wasting. The gait is slightly spastic and slightly disorderly. The knee-jerks are about normal; as a rule the knee-jerks do not remain affected at such a long period after the original trouble. The plantar reflex shows the extensor response. I should call this a mild case of bilateral hemiplegia, or diplegia. There can be no doubt that at some time in the infantile period he had a double lesion of the brain, but of what nature must always be a question.

Hitherto we have discussed only the motor paralysis side of the picture, and it will be admitted it is a sufficiently serious and important one in itself. But unfortunately there are two associated cerebral conditions which invest this infantile disease with a terrible importance, namely, arrest of mental development, or mental degradation, and epilepsy. An extensive lesion of the cortex will almost certainly affect the mental growth of the child, and many of these children are idiots. It is not too much to say that a large number of the examples of so-called congenital idiocy in our asylums belong to this type of disease, for the defective mental condition may persist when the motor paralysis has completely recovered, and in such cases we must assume a remaining lesion of the anterior and posterior lobes of the brain. Unless the lesion is confined to the motor cortex, a cerebral paralysis will nearly always leave its mark on the mental growth of the child, and if the disease occurs at or after the age of two years, when the child has learnt to speak, there is too often a retrograde movement both morally and educationally. The association of epilepsy with this disease constitutes another grave aspect of it. As has been stated, the onset of a cerebral paralysis is very frequently marked by a bout of fits, and the instability of the cortex becomes in many cases confirmed, so that the child grows up not only paralysed but epileptic also. I must remind you of the very large percentage among epileptics whose first fits occur in the first year of life, the so-called "teething convulsions;" it seems not unreasonable to assume that some of these owe their convulsive tendency to a brain lesion of the same nature as that now under consideration, but one in such a situation as to produce no paralytic symptoms.

2. *Paralysis due to degeneration of the cortex.*—I cannot leave the discussion of the cerebral palsies of infancy without mentioning an important group, sharply defined from the two preceding ones, namely, infantile cerebral degeneration. Attention has been drawn to this affection in this country by Drs. Russell and Kingdon * and Dr. Collier. † In traumatic and inflammatory cases we have a lesion sudden, not only non-progressive, but rather with a tendency to recovery. In infantile cerebral degeneration we find a degenerative process affecting the cortex, and in the majority of cases pursuing a relentless course to a fatal issue. As the whole cortex is affected the symptoms are those of motor paralysis with rigidity on both sides of the body; in other words, of spastic diplegia, with also progressive mental degradation and optic atrophy.

* 'Medico-Chirurgical Transactions,' 1897, vol. lxxx, p. 87; and Allbutt's 'System of Medicine,' vol. vii, p. 728.

† 'Brain,' vol. xxii, p. 373.

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A curious point in the ætiology is that the typical disease occurs only in Jews: there is also a tendency for it to appear in several members of the same family. As a rule the child is naturally born, but he is usually one of the later members of a large family, begotten, as has been suggested, when the reproductive powers of the parents are somewhat on the wane. At about the sixth month the symptoms begin to show themselves, and we may divide the course of the disease roughly into three stages. First, a gradual weakness in the muscles, especially those of the neck and back, so that the child can no longer hold up its head; if you try to sit it up, the head lolls over to one side. The eyesight becomes impaired, and there is found a commencing optic atrophy. In the second stage the child becomes apathetic, he no longer takes notice, and he is evidently much affected mentally. The muscular and general weakness are still more marked; and now appears a slight rigidity of the muscles, and the knee-jerks become increased. The rigidity is noticed in all the affected muscles, and its early appearance may be said to be a characteristic of this form of diplegia; the rigidity of the other forms of diplegia is usually a late phenomenon. The third stage may be said to be marked by a definite rigidity of the muscles, so that the child becomes quite stiff the legs are stuck out straight, the feet are extended, the arms are extended, and there is also an over-extension of the hand muscles, so that there is a tendency to claw-hand. Another distinguishing point is the absence of convulsions, which are so common in the other diplegias. The optic atrophy becomes very pronounced towards the end of life, so that the child finally becomes totally blind. Another interesting and, as Dr. Russell considers, a characteristic feature is the condition of the fundus in the region of the macula, namely, a whitish-grey oval patch almost twice the size of the optic disc, in the centre of which may be seen the fovea centralis standing out as a dark red dot. As far as we know at present the disease is invariably fatal in about one and a half to two and a half years.

Morbid anatomy and pathology.—Now I should like to say a few words about the pathology of these diseases.

Birth palsies.—These are due to some injury to the brain at the time of birth. It is conceivable that the brain tissue may be actually lacerated in cases in which much roughness has been used, but the injury is usually a vascular one, and the symptoms are due to hæmorrhage. The situation of the hæmorrhage will, of course, depend upon the vessels injured. As might be expected, the hæmorrhage will be found most often on the outside of the brain.

Two vascular systems are liable to injury, the meningeal and the cerebral. The most important meningeal vessels are the branches of the middle meningeal, and the resulting bleeding will be found either between the dura mater and the skull or between the dura and pia mater, in the form of a hæmatoma pressing on the brain, but not necessarily disorganising its structure. As time goes on the extravasated blood either becomes absorbed, or organised into a membrane, or the formation of a cyst may result.

In the case of injury to the cerebral vessels the immediate consequences may be much more serious. Here, again, the bleeding may take place into the arachnoid space between the dura and pia mater as in meningeal hæmorrhage, or it may track between the pia mater and the cortex. In the latter case the nutrition of the cortex may be gravely disturbed, owing to stretching, rupture, or thrombosis of the small branches of the cerebral arteries which pass at right angles from the supporting pia mater to nourish the convolutions, and there can be little doubt that the most serious cases of birth palsy owe their extensive and abiding symptoms to this cause. I have often been surprised at the small amount of leakage of blood that will prove fatal to adults when in this situation, *i. e.* between the pia and the cortex; quite a thin film will be fatal if of sufficient superficial extent.

Inflammatory lesions.—Now to consider the pathology of our second group of cases, those of disease of the hemispheres due to some lesion of which the nature is yet undetermined,—a lesion sustained once and for all, and not progressive. Here I show you the brain of an idiotic child who died of broncho-pneumonia in St. Bartholomew's Hospital. The patient presented most of the characteristic symptoms of a cerebral diplegia. The brain shows very clearly a long strip of cortical sclerosis about one inch wide running the whole length of the brain from the frontal to

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the posterior regions, and symmetrically placed in both hemispheres. The spirit in which it is preserved has unfortunately destroyed all colour, but on removal from the body the brain was a very striking object. The strips of sclerosis were of a bright yellow colour, which contrasted strongly with the natural colour of the rest of the cortex. They were depressed somewhat below the general level, and the convolutions were much shrunken; that part, for instance, of the ascending parietal convolution included in the sclerosed area is shrunken to about half its normal size. As another result of this shrinkage the sulci are widened, giving the appearance known as "walnut." The sclerosed brain tissue is hard and tough, suggesting by the way it cuts such a condition as cirrhosis of the liver. An enlarged photograph of a section of the affected area shows most of these features well, the grey matter has disappeared except a few islets here and there, and is replaced by fibrous tissue; there are in the centre of the convolution a few white fibres, as shown by the Weigert-Pal staining method, probably connected with the islets of grey matter above alluded to. Microscopical examination shows that the convolutions in these sclerosed areas consist almost entirely of fibrous tissue with many nuclei, and an absence of the normal pyramidal cells of the cortex. [Microphotographs were shown, with those of normal brain for comparison.]

Here is another interesting brain, also from an idiot child with diplegic symptoms. The skull had had two large strips of bone removed over each hemisphere by Mr. Willett, with the hope of allowing the brain to expand. When we look at the brain we see how vain such a hope must be, for we find the same condition of things as in the last case, but in another part of the cortex, namely, both posterior lobes, again symmetrically disposed on either side. The rest of the cortex appears to be fairly natural, but owing to the shrinkage of the posterior lobes the cerebellum is much exposed, instead of being almost hidden as it should be.

As to the pathogenic process which has given rise to the condition above described there is still much debate. Some take the view that the original lesion is of an inflammatory nature, a polio-encephalitis comparable to polio-myelitis of the cord in infantile spinal paralysis. This seems, to my mind, the most probable view. The acute onset, fever, and tendency to regression suggest a germ infection; but of this no proof, so far as I know, as yet exists.

Another view is that the primary pathogenic process is of the nature of vascular occlusion, thrombotic or embolic. We may, I think, admit this only in so far as thrombosis of vessels is an essential step in all inflammatory lesions.

Infantile cerebral degeneration.—The pathogeny of infantile cerebral degeneration is at present involved in obscurity. The condition is one of progressive atrophy of the essential nervous cellular elements of the cortex generally, of which the primary cause must be a matter of speculation. The result is a general wasting and shrinking of the brain, widening of the longitudinal fissure and sulci, and thinning of the individual convolutions.

The "walnut" appearance becomes very marked, and the cerebellum is no longer covered by the overhanging posterior lobes.

Here is a cast of the brain of a child, a patient of Dr. Jackson's, which died in the hospital with spastic diplegia, optic atrophy, and progressive mental degradation. The case is not quite typical of this class of disease, in that the child was not a Jew, and did not present the peculiar appearance of the macula above referred to. Moreover the brain and cerebellum present, in addition to the general atrophy, some patches of sclerosis similar to those in the last two cases. [Photographs shown of patches of sclerosis in cerebellum.] These patches may, of course, be not of inflammatory origin at all, but only local atrophy more extreme than elsewhere.

The microscopical appearances, however, are sufficiently characteristic; and the examination of sections reveals the fact that there is hardly a normal cell in any part of the cortex. I show you here microphotographs of sections made by my colleague, Dr. Collier, one of a convolution of the brain, and another of the normal cortex from a child of the same age, the specimens having been prepared in the same way and at the same time by the Nissl method of staining; the negatives also were taken with the same power, and they have been similarly enlarged, so they have been treated practically identically. You will see that the pyramidal cells, which are such a striking feature in the normal one, with the deeply stained microscopic

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chromatophile granules of Nissl have all gone, and all that remains is a lacuna, and in the lacuna an indefinite mass which takes the stain to some extent, but shows none of the characteristic features of the normal ganglion cell. The disease, then, is essentially an atrophy of the cells of the brain all over, a progressive disease, going on, as far as we know at present, to a fatal termination.

One other point remains to be mentioned. In the whole of the motor pyramidal tracts in the brain and cord we find, by the Marchi staining method, recent degeneration of nerve-fibres. This is further evidence of the gradual progressive nature of the degeneration of the cortical cells, which is the distinguishing feature of this disease. In inflammatory or traumatic lesions such a secondary degeneration must, of course, result if the motor area is involved, but at the time of examination, generally years after, the degenerated fibres have disappeared, and their place is taken by sclerotic tissue.

CERTAIN USES OF ELECTRICITY IN DISEASES OF THE NERVOUS SYSTEM.

By LEONARD S. DUDGEON, M.R.C.P.Lond.,

Assistant in the Electrical Department, St. Thomas's Hospital, Pathologist and Registrar to the East London Hospital for Children.

In these remarks it is intended to describe under two headings the uses of electricity in diseases of the nervous system—(1) the methods in practical use for the diagnosis and prognosis of diseases of the nervous system, and (2) electro-therapeutics in those cases in which it has been found to be useful, either in our experience or in that of competent observers. A brief reference will be made to those essential elements of electricity which are necessary for the proper examination and treatment of neurological cases. The sources of electricity in general use for this purpose are—(1) galvanic, continuous, constant, or battery current; (2) faradic, induced or interrupted current.

Galvanism.—If two pieces of metal, *e. g.* carbon and zinc or copper and zinc, are placed in a solution of dilute acid, the positive electricity accumulates in the copper, the negative in the zinc, and there will be a difference of tension or potential between them. As soon as the metals are connected by a conductor, an electric current will flow in the conductor from copper to zinc, and in the fluid from zinc to copper. This current, which is known as the galvanic current, continues to flow as long as there is an electro-motive force. In a closed cell or circuit the metals are connected by a conducting wire. In the absence of this wire, the term open cell or circuit is applied. The end of the wire attached to the copper plate is called the *anode*, or *positive pole*, *i. e.* current exit; the end attached to the zinc plate is known as the *kathode*, or *negative pole*, *i. e.* where the current re-enters the battery. When the free ends of the wire are held by a patient the current will pass through him from anode to kathode.

Electro-motive Force.—This is the power which disturbs the equilibrium and separates the positive from the negative electricity. The intensity of the electro-motive force is in proportion to the intensity of the chemical action (metals and liquids), but it is quite independent of the size or shape of the metals employed in the battery.

An electric current decomposes the fluids through which it is passing, *e. g.* water is split up into hydrogen and oxygen, so that oxygen is deposited on the negative pole and hydrogen on the positive pole. This action, which leads to considerable diminution in the strength of the current, is termed *polarisation*, and takes place in the battery itself.

Depolarisation.—This can be achieved in various ways, by (1) shaking the metals, (2) chemical action, and (3) blowing air into the fluid.

In order to be able to express in figures the strength of the current and the amount of resistance, etc., certain units have been introduced—(1) a *volt*, the unit of electro-motive force; (2) an *ohm*, the unit of resistance; (3) an *ampère*, the unit of strength of current. In medicine we use a milliampère as the unit, *i. e.* one thousandth of an ampère. A source of electricity with an E.M.F. of one volt, passing through a circuit, the resistance of which is 1000 ohms, produces in it a current of one milliampère.

Now the relation which exists between the electro-motive force, resistance, and current in any circuit is most conveniently expressed by Ohm's law—"The strength of the current in any circuit

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increases with an increase of electro-motive force, and in the same proportion, but decreases equally with the increase of resistance." This is usually expressed by the formula— $\frac{E}{R} = C.$, *i. e.* $\frac{\text{Electro-motive force}}{\text{Resistance}} = \text{Current.}$

Resistance.—Metals, especially silver and copper, are the best conductors, but they vary to a considerable extent.

The electrical resistance of the human body is chiefly in the epidermis. This regulates the amount of current sent by a definite electro-motive force, which shall be transmitted through a given part of the body, but it does not control the diffusion of the current through the deeper tissues. The horny layer of the epidermis, *e. g.* of the heel, offers very great resistance to electric currents, more so than any other part of the human frame. It is therefore easy to see how the electrical resistance varies in different people. Nothing is more obvious than this practical lesson. Some require a very strong electric current to produce any effect, but in others a similar current will cause great agony and violent contractions of the muscles. It is also known that much less resistance is offered by the skin covering the face than by that of the heel. The skin of children and old people often shows a high degree of electrical resistance. We must always remember that the conductive power of the skin is much increased when it is well moistened. The electrical resistance of the skin usually varies between 1000 and 5000 ohms; of course the electrodes and epidermis must be thoroughly moistened with warm water during an examination. Muscular tissue appears to be the best conductor of electricity in the body, therefore some observers use intra-muscular electrodes so as to avoid the peripheral resistance in the epidermis.

Cells.—The chief variety of cell in general use is the Leclanché (dry or liquid). These cells have an E.M.F. of 1·5 volts, and they usually last about eighteen months. Therefore durability is the great advantage of this type of cell, also if a dry cell is upset no damage is done. These cells are arranged "in series" in batteries, generally about thirty to forty in number, *i. e.* an electro-motive force of $40 \times 1{\cdot}5 = 60$ volts.

Galvanometers.—These instruments which record the strength of the current, which passes through the patient, in milliampères, are *absolutely essential* for correct electrical work.

Reversers.—By means of a brass handle on the roof of the battery, we can alter the testing electrode, *e.g.* the negative can be replaced by the positive electrode, and *vice versâ*. This is of great advantage in all neurological examinations.

Electrodes.—The usual varieties which we use at St. Thomas's Hospital are—(1) flat electrode, diameter $\frac{1}{6}$ inch, for the motor points of the facial nerve, etc.; (2) flat electrode, diameter $\frac{1}{4}$ to $\frac{1}{2}$ inch, which is the common type of testing electrodes; (3) large, broad, flat, and indifferent electrode, which is usually applied to the patient's back during most electrical examinations; and (4) various electrodes for therapeutic purposes, which will be referred to later. All these instruments should be covered with chamois leather. Canvas caps to fit over the chamois leather may now be obtained, and these are used only once for each patient. The size of the electrode is of great importance, the larger the electrodes the smaller is the resistance of the human body. As we usually record our observations in milliampères, we should always state the size of the electrode used, because the density of the current can be thus obtained. This varies according to the strength of the current, and the sectional area of the electrode.

Rheostats.—These instruments regulate the strength of the current by means of resistance which is added to the circuit. These are constructed of graphite, liquids, or metal. The first-named is most commonly used.

Alternators.—By this apparatus we are able to use combined faradic and galvanic batteries, and so rapidly change the faradic current which is in connection with the terminals for the galvanic current, and *vice versâ*.

Crank Collectors.—A number of pegs connected with, and equal to the number of cells in the battery, are arranged in a circle, so that a crank can be brought in contact with each of these pegs. No interruptions need be produced, as by modern improvements, a crank touches the next peg before it has quite left the last. If we leave the crank touching two pegs, *e.g.* 8 and 9, at the same time, the ninth cell is short-circuited, no harm will happen at first, but after any length of time the cell will be exhausted. Double collectors are generally

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recommended, as the first row of cells, which are much more frequently used with a single collector, are therefore more rapidly worn out. By means of a double collector the cells of the battery are used equally.

The Determination of the Poles.—(1) When a piece of moistened blue litmus paper is applied to the electrodes, the positive pole is indicated by the litmus paper turning red. (2) Much more marked muscular contraction occurs with the application of the negative pole to a motor nerve in normal people, than with the anode (on closure of the current KCC > ACC). (3) When the electrodes are dipped into a solution of starch and iodide of potassium, a deep blue colour appears around the anode. (4) If we place the electrodes in a basin of water, much more bubbling occurs at the negative pole (hydrogen) than at the positive pole (oxygen).

FARADISM.—When a closed coil of wire is brought into proximity to another closed coil of wire, through which a galvanic current is flowing, similar currents are developed in each coil, but the flow is in the opposite direction in the former. When the coils are rapidly separated an induced current is generated, flowing, however, in the same direction. The current is only produced, however, while the coils are being approximated or separated, not while they are still.

The galvanic current flows through the coil which is known as the primary coil. These coils usually have between 100 and 600 turns of wire. Their resistance usually varies between 5 to 15 ohms. The coil in which the induced current is generated is known as the secondary coil. This is generally constructed with a large number of turns of wire, about 1000 to 5000, as it is usually necessary to obtain a high E.M.F. The resistance of the secondary coil varies between 100 and 900 ohms (*see* below).

The electro-motive force of the induced currents depends on—(1) the greater the number of turns of wire on a coil, the higher is the E.M.F.; (2)

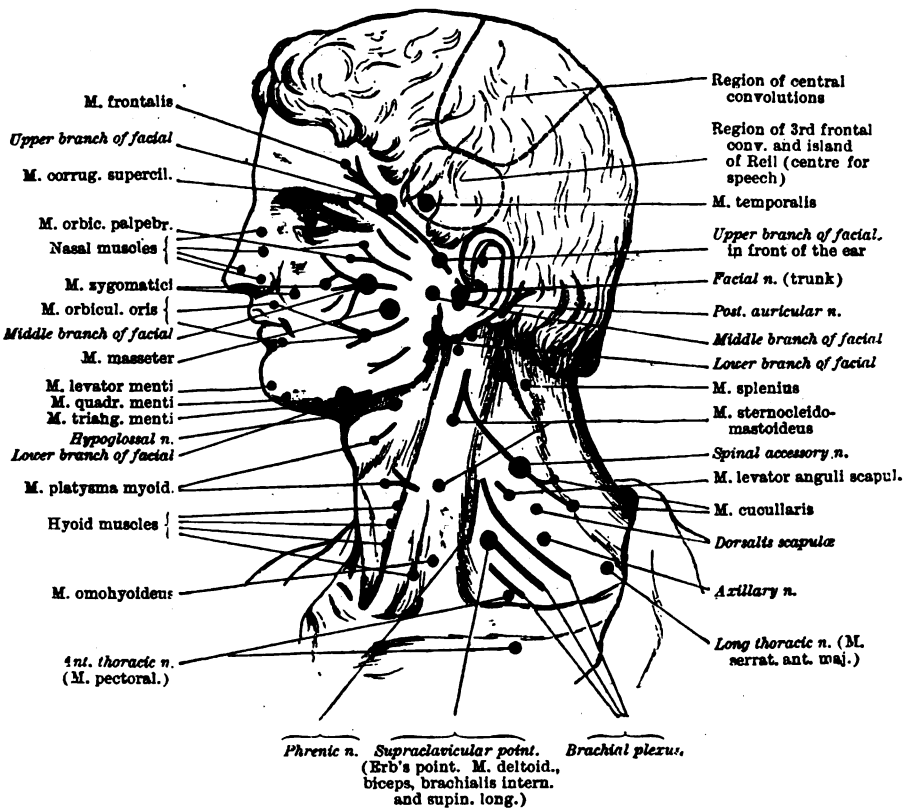

FIG. 1.

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the stronger the inducing current, the higher is the E.M.F. of this current; (3) the presence of an electro-magnet also increases the E.M.F.; (4) the suddenness of the break of the inducing current.

The inducing power of an electric current is much increased if it acts simultaneously with a magnet. This is arranged as follows:—The primary wire, *i.e.* the wire through which the inducing current passes, is wound round a cylinder into which an iron core can be adjusted. This core should be made of a number of soft iron wires, as these are able to take and loose magnetism more quickly than solid iron. When this core is introduced into the centre of the primary coil, and as soon as the current passes, the iron core is converted into a magnet, and by this means the strength of the induced current is much increased. The strength of these currents depends on Ohm's law.

What is the primary current? If a galvanic cell is allowed to discharge through a bobbin of wire, and the circuit is completed by means of an interrupter, so as to make and break the current automatically, an extra current is induced in these parallel spirals. If this current is allowed to pass through a patient it is called the primary current.

The faradic current produced in the secondary coil is called the secondary or alternating current.

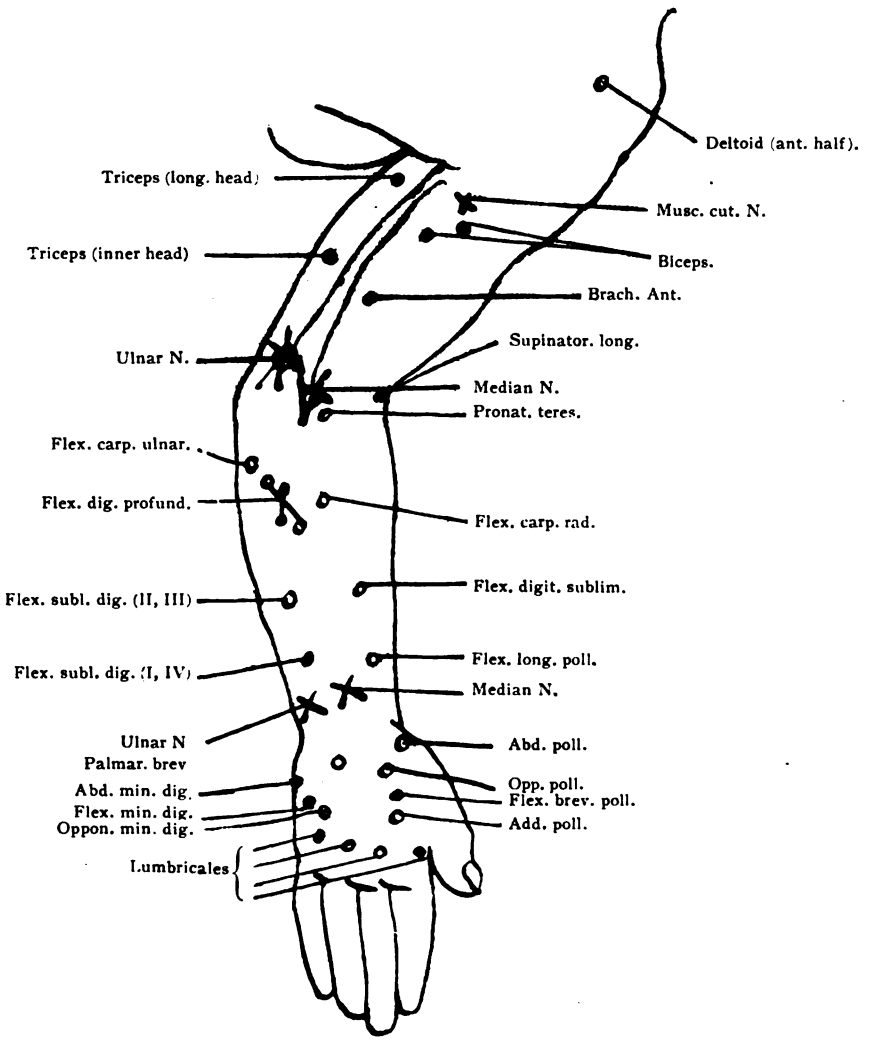

FIG. 2.—Motor points; front of arm (Erb.).

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The strength of this current is usually regulated by the sledge of Dubois-Reymond. The approximation of the primary and secondary coils increase the strength of the current, and *vice versâ*. The effects produced by it, *i. e.* the secondary current, depend considerably on the diameter of the wire which is used. If very fine wire is used, only slight muscular contractions occur, but there is generally marked local pain; as we increase the diameter of the wire, the muscular contractions become more powerful; if the secondary coils are wound with thick wire such as is used for the primary coils, a similar effect will be produced, *i. e.* strong local, or perhaps even diffuse muscular contractions similar to those obtained with the primary current.

The inducing current is made and broken by means of an electro-magnet and a spring. When the primary current is allowed to pass through the electro-magnet, the latter becomes magnetic, and attracts the vibrating hammer (Wagner's), which therefore leaves the platinum point of the contact screw; this interrupts the current, the magnet ceases to be magnetic, and the attached spring causes the hammer to fly back; the process is repeated as rapidly as desired. The interruptions are regulated by a contact screw, which controls the movements of the vibrating hammer by means of a spring, *e. g.* the interruptions become more rapid the further we screw the contact screw home, and *vice versâ*, but only up to a certain point; if the spring is screwed down too tightly, the hammer cannot vibrate, and is therefore rendered useless. The strength of the spring and of the inducing current have important bearings on the rapidity of the interruptions. A piece of

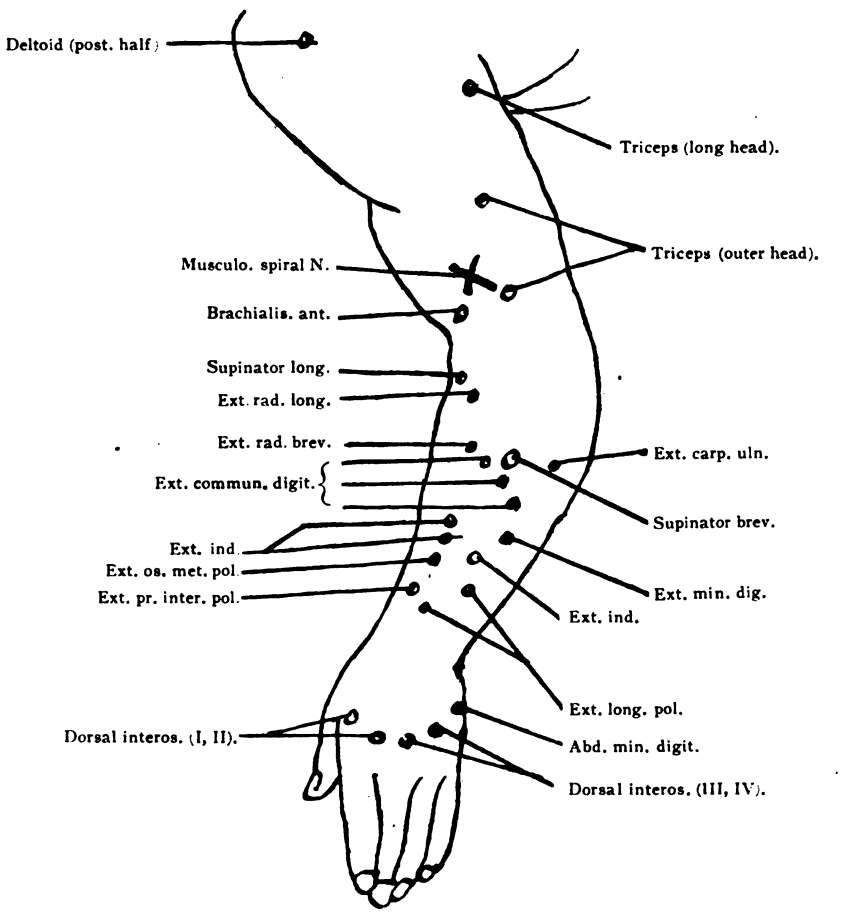

FIG. 3.—Motor points; back of arm (Erb.).

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wire on which slides an aluminium ball can be attached to the vibrating hammer; by this means the vibrations are rendered slower according to the height of the ball on the perpendicular side of the bent wire. The muscular contractions become more powerful and painful the slower we make the interruptions.

The motor points.—An accurate knowledge of the position of these points (Figs. 1 to 6), where motor nerves and muscles are most easily excited, is absolutely essential. The difference which it makes can be appreciated by any one, whether the testing electrode is placed on a motor point or in its immediate neighbourhood. These points do vary in position in different individuals, but not to any very great extent. Therefore, if we wish to obtain the position of a known motor point, we place the electrode in the position which this point should normally occupy, and continue to move the electrode in the neighbourhood of this point until the greatest muscular contraction is obtained.

Practical testing.—It is advisable to commence with the weakest interrupted current which will produce contraction of the muscles. For example, suppose the patient under examination is suffering from ulnar paralysis. We * first fix a large flat electrode to the patient's back, then apply the testing electrode to the different motor points of the ulnar nerve in the arm and forearm, the weakest current is recorded which produces contraction in the ulnar muscles. The normal arm is also tested, and the difference in the reactions carefully noted. It is important always to commence with a weak current, and always to compare the normal with the abnormal. The reason for laying such stress on these points is—(1) patients vary considerably in their excitability to electric currents; (2) there is no need to use a stronger current than is required to produce a definite contraction in the muscles.

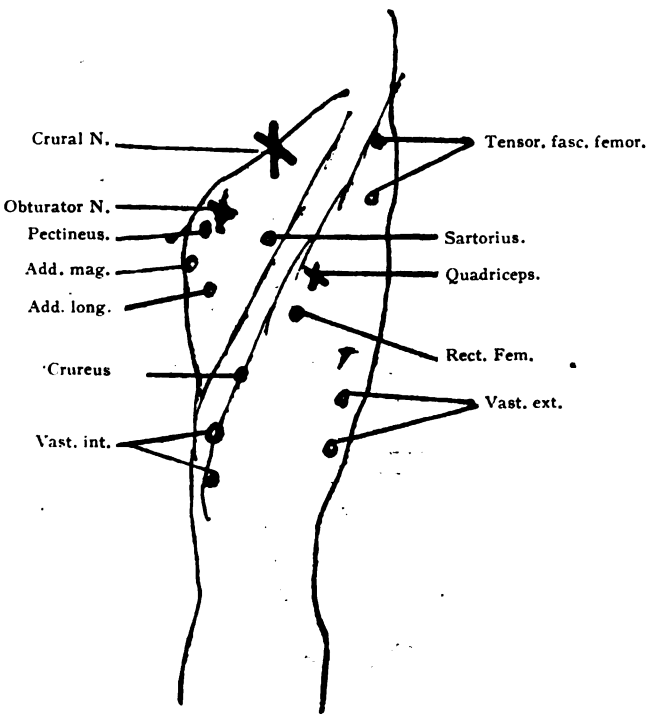

FIG. 4.—Motor points; front of thigh (Erb).

It is necessary to test the electrical sensation of the skin most carefully while examining the deeper structures, and to compare the result in the affected region with that in the normal. Patients

* In all electrical examinations it is absolutely essential that both the electrodes and the patient's skin should be thoroughly moistened with warm water.

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who are very fat generally require a stronger current and a larger electrode than thin people. When testing muscular contractions, either by the faradic or the galvanic current, we should always *feel* the contractions as well as inspect them.

After full examination with the interrupted current, the next observation must be made with the continuous current. We should commence with the kathode as the testing or active electrode, and then complete the examination with the anode, by reversing the poles.

Here again, the weakest current which will produce well-marked muscular contraction is all that is required. It is generally advisable to commence with a current of 3 ma. and to increase the strength if necessary. The contractions must be noticed both at make and break with the negative and positive electrodes. The character of the muscular contraction, whether sharp or sluggish, must also be noted.

What is meant by normal electrical reactions? If, when a weak faradic current is applied to the motor point of a peripheral nerve, it produces a sharp contraction of the muscles supplied by that nerve, and the contraction is equal on the two sides of the body, the conclusion would be justified that the electrical reactions of the nerve in question were normal. If the muscles supplied by that particular nerve are also themselves tested with the faradic current * and react in the same way, and with a weak galvanic current give a short, sharp contraction which is equal on the two sides of the body, as registered by the galvanometer, and the contraction shows any of the following polar formulæ :

(1) KCC > ACC > AOC > KOC ; †
(2) KCC > AOC > ACC > KOC ;
(3) KCC > AOC = ACC > KOC ;

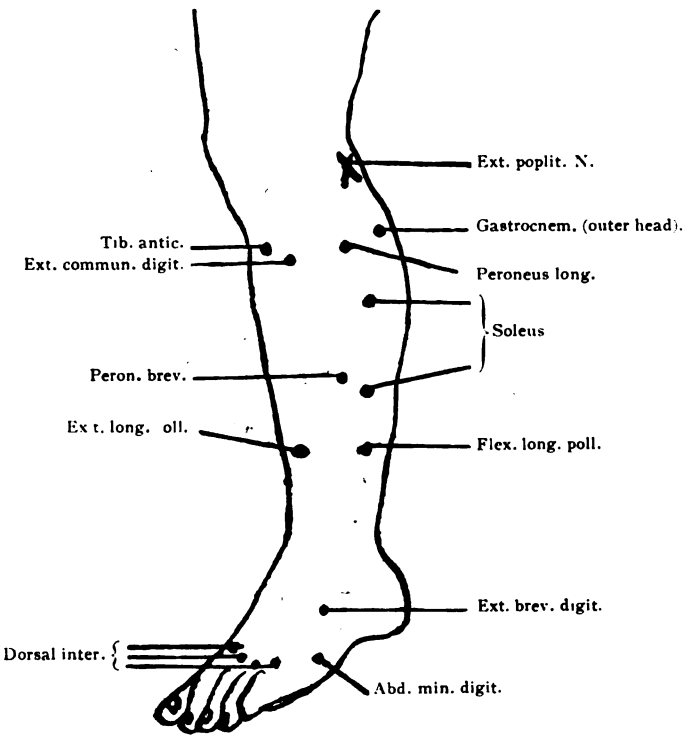

FIG. 5.—Motor points ; front of leg (Erb).

we should know that the electrical reactions of the muscles in question were normal.

What is meant by the polar change? When we apply an electrode to a normal muscle, the

* Von Ziemssen has found that "the points" where muscles can be most easily made to contract by direct stimulation correspond exactly to the spot where motor nerves enter the muscles. The direct faradic muscular excitation is therefore always a nerve excitation. Von Ziemssen, quoted by Strümpell, 'Text-book of Medicine,' third American edition, 1901, p. 842.

† W. S. Hedley, 'Therapeutic Electricity,' 1899, p. 112.

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galvanic current produces a muscular contraction both at make and break, and if the electrode which we use is the negative and active electrode, it will be noticed that a stronger and sharper contraction will occur on closing the current (KCC) than with the positive electrode (ACC). The contractions produced both by opening and closing the circuit, with the positive electrode, may be greater than, equal to, or less than each other; but the contractions produced on closing the current with the negative electrode are always the greatest in the normal, while in very marked cases there may be very feeble contractions on faradic stimulation, or a very strong current is required to produce the contractions. The excitability of the nerve may not be completely lost, or only slightly diminished, yet the galvanic changes may be characteristic of those met with in complete R.D., viz. inversion of the formula, with sluggish and exaggerated contraction in the muscles supplied by the nerve in question. In fact, partial reaction of degeneration is very commonly obtained, *far more*

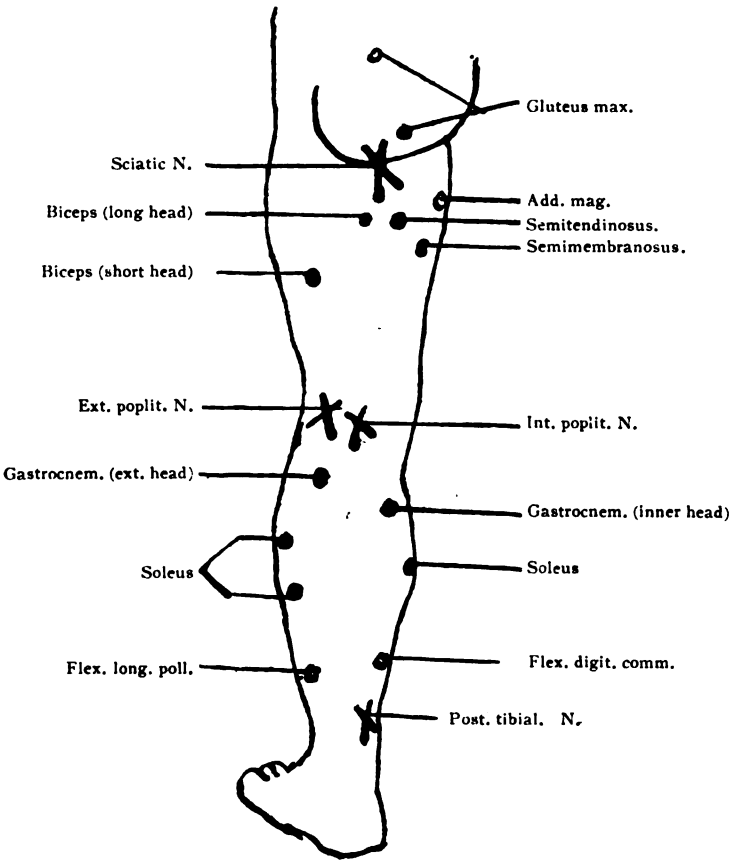

FIG. 6.—Motor points; back view of leg (Erb.).

health.* The first-named is the usual formula in healthy muscles.

What is partial reaction of degeneration? It will be found by faradism that it requires a stronger current to produce muscular contractions as compared with the normal side, and that the contractions are feeble, or at any rate less powerful than *so* than the complete R.D. of Erb. Partial reaction of degeneration may be anything short of complete R.D.

What is meant by complete R.D.? Professor Erb,* who was the first to describe the condition known as "The Reaction of Degeneration," summarised it as follows:—"(1) Abolition of

* ACC > KCC > AOC > KOC is described as occurring in apparently healthy people. If so, it must be extremely rare.

* Professor Erb, von Ziemssen's 'Handbook of General Therapeutics,' 1887. English translation by A. de Watteville.

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faradic excitability in nerve and muscle; (2) exaggeration of galvanic excitability of muscle; (3) a sluggish contraction of muscle; (4) inversion of the normal formula, *i. e.* ACC > KCC, and this is followed by KOC = or > AOC." That is to say, there is no response of the nerve or muscles to the faradic current, no response on galvanic stimulation of the nerve; but the affected muscles show the well-marked changes recorded above. This same authority also calls attention to the fact which he considers most important in the reaction of degeneration, viz. "a slow and prolonged contraction with a very weak current is always present, and may be regarded as pathognomonic of the condition."

How soon after the division of a nerve are electrical changes obtained? Reference must be again made to Professor Erb* concerning this most important point. A case with a paralytic lesion of a motor nerve, occasionally shows a slight increase of electrical excitability lasting about a day or two; but as a rule, immediately or within three days after the paralytic lesion, a progressive uniform lowering of galvanic and faradic excitability occurs in the affected nerve. The excitability is seen to diminish more and more, so that at the end of seven or ten days a very strong faradic or galvanic current may fail to elicit the slightest contraction.† The defect begins in that part of the nerve nearest to the lesion, and spreads rapidly to the periphery. The first signs of returning excitability appear at the same time for both currents; they are first seen in the central end of the nerve, and slowly travel towards the periphery. It is very important to remember that voluntary power is *frequently* restored, before the electrical reactions return to normal. Professor Duchenne, who was the first to notice this fact, laid great stress upon it. At the present time there are two cases of facial paralysis attending the electrical department at St. Thomas's Hospital; the paralysis has almost completely recovered in each case, but the electrical reactions are still severely affected. This simply shows that the cut nerve can conduct some impulses much better than others.

Faradic Current.—The reaction of the muscles is almost the same as of the motor nerves, a constant decrease of excitability takes place, proceeding, in the course of the second week, to its total disappearance.

Galvanic Current.—During the first week a gradual loss of galvanic excitability takes place in the affected muscles; but about the end of the second week this diminution is replaced by an increase of galvanic excitability, which, within the next few weeks, may reach a very high degree of exaltation, and exhibit all the changes mentioned previously. This peculiarly characteristic change in the muscles persists without further change for three, six, or even eight weeks. This change is followed by a gradual diminution of galvanic excitability, while the qualitative modification, especially the sluggish contraction, continues to be present. In incurable cases this diminution progresses, and it becomes more and more difficult to excite the affected muscles. The polar change, ACC > KCC, no longer exists, and only a very feeble ACC remains. It may be a question of years before the galvanic excitability is quite lost in the degenerated muscles. In cases that recover, with the return of voluntary power the electrical excitability and the other normal qualities of the muscles are gradually restored in proportion to the rate of repair, but, as before mentioned, the power which muscles possess to contract to electrical stimuli is generally much slower to return to the normal, than motor function. It is found that nerves recover their power of conducting electrical impulses more rapidly than muscles; this is due to the more severe morbid changes produced in the latter.

How does the presence of the reaction of degeneration influence the diagnosis? This shows that there is, or has been, degenerative atrophy in the nerves and muscles of the affected part; also that there is a lesion, either of (1) peripheral nerves, (2) motor roots, or (3) central grey substance (anterior horn cells); and absolutely excludes a cerebral lesion. The whole importance of the reaction of degeneration is due to the fact that we are able to obtain an absolute diagnosis between diseases of the upper motor segment from those of the lower. But it does not differentiate all diseases of the lower motor segment; far from it, it is very often of no value for such purposes.

What is the value of R.D. as a prognostic sign?

* Ibid.

† In some cases complete loss of electrical conduction in the cut nerve takes place within forty-eight hours.

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It is generally stated in text-books of medicine that the partial reaction of degeneration is of better prognosis than the complete. This may be so, but I cannot see on what basis such a statement rests. The complete reaction of degeneration, as described by Professor Erb, is by no means of common occurrence, while partial R.D. is very commonly met with, and frequently in exactly similar cases to those in which complete R.D. has been described. I am *certain* partial R.D. has frequently as much bearing on a case as complete R.D. Dr. Lewis Jones* substantiates what I have already mentioned.

"The polar change, with predominance of ACC over KCC, is referred to by Professor Erb as one of the most constant phenomena in medicine, but, in my experience, and in that of others, its appearance is so inconstant as to be absolutely without diagnostic value as a part of the reaction of degeneration. Again, the degree of sluggishness, or, in other words, the duration of the contraction produced in muscles by the closure of the current from the cells, varies within wide limits."

Prognosis of diseases of the lower motor segment has been discussed in detail by Professor Strümpell.† His statements are based on the changes met with in electrical reactions, chiefly in relation to the seventh nerve.

The Mild Form of Facial Paralysis.—Electrical excitability in the facial nerve and paralysed muscles remains entirely normal. Strümpell considers that recovery is rapid, usually in two or three weeks.

The Middle Form of Facial Paralysis.—In this case there is only partial R.D. The excitability of the nerve is diminished, but not lost. In the muscles, however, in about two or three weeks the usual changes met with in complete R.D. appear. Here, according to Strümpell, the prognosis is good. Recovery takes place in about four to six weeks.

Severe Form of Facial Paralysis.—There is complete R.D. Strümpell says that recovery is slow, if it occurs at all; only after three to six months, or even later.

Anybody who gives a prognosis which is in agreement with the above facts must often fail to please either the patient or himself. Frequently cases in which only partial R.D. is present take from six months to a year to recover, and even then, sometimes no improvement takes place, but yet the complete R.D. of Erb will never occur. Complete R.D. is often stated to be present in cases where a careful electrical examination has never been made. Again, what some people term as complete R.D. is really incomplete, but the former is the usual term employed. I give below two excellent examples which show that the electrical reactions are only of value when considered relatively, and when balanced with the other physical signs obtained.

1. A girl æt. 22. Facial paralysis following "cold." For the last six months her electrical reactions have been partial R.D., and have been examined every month; the girl still has very marked facial paralysis, in fact almost as bad as when she was first seen seven months ago.

2. A woman æt. 34, with paralysis of both the seventh and eighth nerves of peripheral origin, following syphilis. It is now nine months since the onset; she has the most marked facial paralysis it is possible to obtain, yet she has never had complete R.D.

Both these cases illustrate the important bearing which partial R.D. has on diseases of the lower motor segment. I will go through with you a summary of the electrical reactions in agreement with our observations:—(1) Complete reaction of degeneration as described by Professor Erb is rare. (2) Partial R.D., which is so very frequently obtained in diseases of the lower motor segment, far more so than complete R.D., proves that its significance is frequently as great as complete R.D. (3) The manner in which a nerve reacts to electric stimulation is of more value in a case with partial R.D. than the condition of the muscles. (4) That muscles which show partial or complete R.D. may have recovered their motor function before there is any improvement in their electrical reactions. (5) That complete or partial R.D. is only of relative value in the prognosis of a given case, and not of absolute importance. (6) Muscles which show no signs of paralysis may yet show a well-marked reaction of degeneration.

(*To be continued.*)

* H. Lewis Jones, 'Medical Electricity,' 1900.

† Professor Strümpell, 'Text-book of Medicine,' third American edition, 1901, p. 861.

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* *Specially reported for The Clinical Journal. Revised by the Author.*

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WITH SIR DYCE DUCKWORTH AT ST. BARTHOLOMEW'S HOSPITAL.

MAY 16th, 1902.

GENTLEMEN,—I shall consider with you to-day the patient who has been brought down here, and whose case presents some features of interest. The name of his disorder you will find in books under the head of *arhythmia cordis*, which signifies irregular rhythmic action of the heart. The peculiarity in this boy is that his heart is very often in a state of hurry. At other times his heart settles down and passes from a state of extreme hurry to one of extreme slowness. His history is very simple. He is sixteen years of age, an errand-boy by occupation, accustomed to be active, and is otherwise healthy. He was fairly well up to two months ago, when he complained of pain in his left side, under his heart, coupled with palpitation. He came to the hospital, received medicine, and had some local applications. He kept quiet for a time, but still suffered from this agitation. One day he felt so ill that he came here, and was admitted (February 21st). He had always been a healthy boy, but sometimes had pain in his heart when he walked quickly or ran, or over-excited himself, and then the heart began to beat quickly. He once had a severe attack of epistaxis. He first noticed this palpitation five years ago, when he was eleven years of age, and at that time attended this hospital on account of it. In 1899 his heart was in such a condition that he was detained in the surgery for twenty-four hours. In May, last year, he came into Rahere Ward for three weeks. On the day he came in he was able to walk rather briskly to the hospital, and without much loss of breath. The family history is very good. His parents are young and are both living, and he is the oldest of six children, all of whom are living

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and healthy. That, of course, is a very satisfactory history with reference to the family. He is a well-grown youth for his age. When he came in his pulse could not be counted at the wrist because of its frequency. The respiratory movements were increased to thirty-two, but there were no physical signs of any moment except those of the heart. The heart was beating in the fifth and also partly in the sixth interspaces just without the left nipple line. His pulse on admission was 212, ascertained by auscultating the heart. He complained of some pain and tenderness in the lumbar region, and the liver was enlarged, extending to two finger-breadths below the cartilages. He was put to bed and kept quiet. His urine was found to be perfectly healthy. The pulse continued to be exceedingly rapid. After he had been in bed for a fortnight he had some disturbance of his stomach, vomiting came on, and he became a little jaundiced; the face also was somewhat puffy, as were his feet and loins. There was discomfort, but not acute distress from that. The pulse-rate later was found to be 154. He slept all day, and was very wakeful at night. He was relieved of the discomfort which I have mentioned by the application of three leeches over the epigastrium.

After he had been in the hospital about a month there was an extraordinary amount of urine passed; on one occasion it had been 22 oz., and on another occasion 27 oz., but it rose up to 165 oz. in a day, with a specific gravity of 1012. This sudden rise in the quantity of urine passed could not be accounted for by anything he had taken; no medicine had produced it; there had been no alteration in his treatment or diet. This great urination was accompanied by disappearance of the œdema in the loins and legs and face. He also slept much better after it. But the most extraordinary alteration in the patient's signs which accompanied the increased output of urine was the sudden fall in the pulse-rate. In two days it fell to 92, after having been 176, 160, 168. The following day, however, the rapid rate was resumed, and it rose to 184. A few days later it fell again, and became 100. Five days later it rose again, and so it alternated until we come to April 10th, when his pulse-rate was the highest which has been noted, namely 224 per minute, and the respirations 32, or a ratio of one to seven. As a rule his pulse is now exceedingly rapid, and he has had a great deal more heart hurry than heart slowness. As you know, heart slowness is called bradycardia, and heart hurry is called tachycardia. This boy has presented both those conditions. His general health all through has been fairly good; his appetite has been satisfactory, and now there are very few physical signs about him except this extreme irritability of his heart, and indications of general hypertrophy of it. The transverse cardiac dulness measures nearly five inches. Yesterday it was determined to try a treatment which has sometimes been effectual in slowing down the pulse, namely, the simple plan of taking a series of deep inspirations. At the end of such a series the pulse is often found to be reduced in frequency. While the boy lay quietly in bed the heart-beats were 192 per minute, and the respirations 28. After five deep inspirations, after each of which the breath was held, not only did his pulse not become slower, but it became more frequent, going up to 216, and the respirations increased to 36. Another attitude was then tried, that suggested by Professor Clifford Allbutt. He recommends, as a means of checking this violent action of the heart, that the patient should bend himself down deeply, so that the knees come in contact with the thorax, taking, at the same time, a series of deep breaths. That was practised in this case, with the result that the pulse rose to 208, though the respirations were not increased in frequency. Therefore the methods which have sometimes been found useful in the treatment of heart hurry have not been of any avail in this case.

We have here, then, a marked example of disorderly action of the heart, or arhythmia; but with this there is little else to note. Meantime the boy is unfit for exertion or exercise, or, indeed, for any active purpose in life. The questions which arise are, where is the seat of this disorder, and what is the cause of it? It is not easy to count any pulse which is over 160 per minute at the wrist. Of course, one has always to consider the element of emotion when one brings a patient into a theatre like this to face an audience, but we generally find that if patients have been in hospital for some time they become accustomed to inspection and being made the objects of demonstration. I cannot now count his pulse at the wrist. The beating of his heart may be exactly likened to the noise made by

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the propeller of an electric launch when it is going at full speed. The first and second sounds of his heart are exactly alike; you cannot tell which is the first and which is the second. The present rate is about 200 per minute. There cannot be much doubt as to the mechanism at fault in a case like this. The fault lies in the central nervous system, or in the nervous supply to the heart. This supply is regulated mainly by two sources, the one which holds the action in check, and the other which encourages it; one acting like a brake and the other stimulating it. The vagus is the channel by which the brake is put on the heart, and if the vagi act too much the heart is too much controlled. The quickening action comes through the accelerator nerves, and if those are called into over-action there is heart hurry in spite of the brake-action of the vagi.

Direct experiments on the heart show that when the action of the vagus is controlled by atropine this inhibitory control over the heart is lost. But even under such experimental conditions the heart-rate is still apt to reach 120 per minute. But you see this boy's heart is acting at nearly twice that rate, so there must be something more than mere arrest of inhibition of the brake-action of the vagi. In many of these cases the patients have faults of both the vagi and the accelerator nerves. The vagi do too little and the accelerators do too much, and between them the disorder is intensified. The question to determine—and it is not by any means easy of determination—is whether the mischief is central, or whether it is in the nerves or in their terminations in the heart walls. This boy has been in a gymnasium and has been accustomed to use his muscles rather violently, and in other ways we may suppose that he exposed himself to some strain, and that the heart has become irritated. But many lads of sixteen years of age strain their hearts every day. Most schoolboys strain their hearts, and no harm comes of it in many cases. They have paper-chases, football tussles, and the like, all of which are very trying to the heart in growing youths. Nineteen out of twenty youths come through the ordeal with sound hearts, but the twentieth boy suffers. He is not made like other boys; there is some fault or flaw in him which tells upon him, and his heart gives out or breaks down. There is probably some fault in the nervous system, central or peripheral, so that it fails to fulfil its functions, and there ensues this irregularity of the heart. There is generally some lesion or sign associated with the abnormality, as there is here in the shape of dilatation. Persistent over-action leads to dilatation, and that is commonly the first thing to appear. There is no indication here of any valvular disease whatever; the respiration is seemingly quiet, and yet the pulse is beating 200 and upwards. The respiration is 32, or about twice the normal number in a youth. If a person suffers from dyspnœa, the result of heart disease, you know how soon he is put out of breath by a little exertion. If a man with a dilated heart runs five or six yards he puffs and blows, and he does the same if he climbs a few steps; but nothing of that sort is taking place here in association with this extremely rapid action of the heart. You know that in pneumonia the pulse-respiration ratio is altered materially; it is about one to two; there is both rapid respiration and rapid action of the heart, the heart only acting twice as fast as the lungs. In regard to treatment, we found tincture of nux vomica was very useful to him, and that seemed to be the best drug we employed in the case. Strychnine and nux vomica are medicines which would naturally affect the accelerator nerves. Belladonna is a drug which would act more particularly upon the vagus. But this patient has had too little reduced action of the heart to warrant any special treatment directed to that phase. The curious flow of urine in the case which I have mentioned would indicate a nervous state, and would point to the nervous centres being involved. Such polyuria has been noted by Allbutt to attend paroxysmal attacks of tachycardia. Probably we should find that any morbid anatomy of these structures would be quite unrecognisable to the naked eye.

In cases of neurosis—and this is, I think, a neurosis—the morbid anatomy is very minute indeed. In cases of insanity, for instance, though there is nothing to show to the naked eye, pathologists, by means of new methods, now show us changes in the brain; and so I have no doubt that in some of these neurotic conditions where the naked-eye pathology shows nothing, the minute anatomy would show definite molecular changes in certain parts which are beyond the limits of ordinary vision. The outlook in these cases of

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rapid pulse and slow action of the heart is not, as a rule, good. One feels anxiety for patients having these signs, for they seldom recover soundly, and generally drift into poor health and die prematurely. We do not know whether the tachycardia or the bradycardia is the worse, but anyone presenting these conditions is not a subject for longevity. Certain people are born with an infrequent action of the heart. The great Napoleon is said to have had a pulse of 40 or thereabout. There are people who live a full term of life with a very infrequent pulse; still, there is often something "uncanny" about them. I remember a saying of the late Sir William Savory, namely, "All eccentricities are grave;" and that is a matter which you will have to consider when you are examining people, especially for life assurance. Any abnormality in any organ which is presented must be noted, and you must be watchful over people who are the subjects of marked bodily or functional eccentricities.

(Since the delivery of these remarks the patient has continued in the same condition with persistent tachycardia. He still derives benefit from tincture of nux vomica in ℥xv doses thrice daily, and no other drug has had any marked influence in reducing the rapid action of the heart. The polyuria has not occurred again.)

CONVULSIVE DISORDERS IN INFANCY.

A Clinical Lecture delivered at the Hospital for Sick Children, Great Ormond Street.

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GENTLEMEN,—The subject which I propose to consider in this lecture is convulsive disorders in infancy; and I use the term "convulsive disorders" advisedly, to comprehend not only the different grades, if I may so say, of general convulsion, but also certain local affections which, although not usually considered under this head, are nevertheless convulsive in character. A proper appreciation of the relation between these various convulsive manifestations has a very practical value both in prognosis and in treatment, the two points which I wish specially to consider.

As our starting-point we may take what is ordinarily recognised as an infantile convulsion. I need hardly describe such an attack, you are all familiar with it: an infant suddenly becomes livid about the lips and about the alæ nasi; its eyes are fixed in a vacant stare, consciousness is lost, the limbs stiffen for a moment, and then face, arms, and legs twitch irregularly, and the eyes perhaps show a nystagmoid jerking to one side or the other, or perhaps a transient squint; at the same time the fontanelle is bulged, and the child becomes more and more livid. Gradually twitching and rigidity subside, consciousness is regained, and the child lies back pale and exhausted. Such an attack may occur as an isolated manifestation, to recur perhaps after weeks or months, perhaps never; but, on the other hand, it may form part of a series of convulsive attacks, between which the infant may not even regain consciousness, and is, in fact, in a condition exactly resembling the status epilepticus of the adult.

The seriousness of such attacks as these no one is likely to overlook, but there are other minor manifestations which I venture to think are often overlooked, or, if not overlooked, regarded too lightly by the medical man. Those of you who have attended the out-patient department here must have heard mothers talk of "the inward convulsions,"—a vague complaint, I admit, but one to which the mother attaches no little importance. Now I shall put in a plea for these "inward convulsions." I would strongly advise you never to disregard such a complaint from a mother; the symptoms to which she refers may be the first sign of coming danger, and may give a valuable indication for the prevention which is better than cure.

As I have said, it is a vague complaint, and the symptoms which the mother includes under this term are not the same in all cases. In one case the child screams, goes a little livid around the mouth, and then perhaps turns its eyes upwards, and at the same time turns its thumbs in strongly towards the palms. In another there is a momentary twitch, extremely fine it may be, of the forearm or of one leg, or perhaps only of the fingers, with no apparent loss of consciousness, and, indeed, with so little disturbance that only a care-

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ful observer would notice that anything unusual had occurred. In a third there will be a sudden start of the whole body as if from an electric shock, so momentary that if it occurs during sleep, as it sometimes does, it scarcely disturbs the infant, who perhaps cries slightly after it, but is soon sleeping quietly again. Trivial as these symptoms may appear in themselves, there can be no doubt that they are just as truly convulsive as the major attacks which we call infantile convulsions; they stand, indeed, in much the same relation to these major manifestations that petit mal does to major epilepsy, and at any moment they may run on into the more general convulsion.

Sometimes, however, the mother's phrase is less accurate, and she applies it to the paroxysmal screaming of an infant with flatulence and colic; unless, indeed, she dignifies that condition by another term from the maternal vocabulary, "the screaming convulsions," which may have no justification in pathology, but may serve to remind us that the same colic which gives rise to the paroxysms of screaming may also give rise to actual convulsions.

In all these conditions, whether the convulsive manifestation be of the major or of the minor variety, there is a common basis in an abnormal nervous instability, and on this I would lay some stress, for it forms a link between these and the other convulsive conditions to which I shall refer. It would seem that this nervous instability may be congenital or acquired; in one case an infant shows a tendency to convulsions almost from the day of its birth, in another it is only in later infancy, when rickets has had time to develop, that a convulsive tendency appears. This difference may be of some prognostic significance; at any rate, I strongly suspect that the infant who suffers repeatedly with convulsions under the age of six months is more likely to show evidence of nervous instability in convulsions or otherwise in later years than is the infant who only develops its convulsive attacks under stress of rickets; though, of course, it must be understood that the child who has already shown the congenital tendency will be specially prone to show its nervous instability even more markedly when the influence of rickets is superadded. Certain it is that there are children who from earliest infancy show a nervous instability which remains for years, and may manifest itself in convulsions at the onset of every acute illness which befalls the child. I would not recommend you to prophesy in such a case, but I remember one case in which the family doctor gained no little credit from having foretold such an occurrence; he had seen the child at the age of three months, when it had had repeated convulsions, and recognising the unstable nervous equilibrium of the child, he told the mother that if ever the child had an acute illness it would probably begin with a convulsion. Two years later, when I saw the child, it had just begun an acute bronchitis with a temperature of 103·8°, and a convulsion.

There can, I think, be little doubt that in certain cases also infantile convulsions foreshadow other manifestations of nervous instability which appear in later childhood; for instance, a boy of ten years had convulsions from early infancy up to the age of three years, since then he has suffered with night terrors, and occasionally walks in his sleep; he is extremely "nervous," and after any little excitement suffers with insomnia. In another child convulsions occurred occasionally up to the age of three and a half years, and at the age of six years he came to my out-patients with a marked habit-spasm, twitching of the facial muscles, and blinking of the eyes. Another child who had suffered with convulsions in infancy came under my care for hysterical dyspnœa at the age of twelve years.

Of course we must be careful in tracing any relation between conditions so common as infantile convulsions on the one hand, and habit-spasm, night terrors, or the various neuroses of childhood on the other; but the sequence is at any rate sufficiently frequent to justify the suspicion that in these cases both are dependent on one underlying cause, a congenital nervous instability, and to this extent the earlier manifestations may foreshadow the later. Dr. Coutts,* in a most interesting paper on the subject, has pointed out that infantile convulsions are often followed by neuroses, not only in childhood but also in later life.

But when we come to consider the convulsive conditions which are associated with rickets it is evident that here we have to do with an acquired morbid state; it is only after the development of

* 'Trans. Internat. Med. Congress,' 1887.

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the rickets that the convulsive tendency appears, and it passes off as the rickety condition improves. And in this connection I should like to draw your attention to an interesting point, namely, that the nervous symptoms of rickets are not necessarily proportionate to the bony changes present; one child with extreme rickety deformity of skull, thorax, and limbs will show no tendency to convulsive manifestations, whilst another with comparatively slight rickety change in the bones will suffer from almost all the possible nervous symptoms of rickets. It would seem, indeed, that, as has been noticed in many other diseases, the stress of rickets may fall upon different tissues in different children; and I have seen cases which strongly suggest that in certain families, as well as in certain individuals, there is, if I may so say, a tissue proclivity, which determines the incidence of rickets on certain tissues. However this may be, I would have you remember that with slight evidence of rickets in the bones you may find marked nervous symptoms; and *vice versâ*, with severe bony change there may be little or no nervous disorder.

The acquired nervous instability of rickets may manifest itself in local as well as in general symptoms, and I wish to lay stress particularly on these local manifestations, for a proper appreciation of their significance has a very practical value.

Laryngismus stridulus is one of these; most of you must have seen, perhaps I ought to say heard, this condition in the out-patient room here; a rickety infant on being awakened from sleep, or perhaps more often still when it begins to cry, suddenly seems to hold its breath, its chest is fixed, its face becomes more and more cyanosed, its features are contorted as if in terror, and just as asphyxia seems imminent inspiration occurs with a long-drawn crowing sound very like the whoop of pertussis. Now what has happened? Some slight stimulus, perhaps a deeper inspiration than usual bringing cold air in contact with the glottis, has started a spasmodic or convulsive closure of the glottis,—a tonic convulsion, in fact, of that particular part. And the close relation of these attacks to general convulsions is seen in the history of many of these cases, in a large proportion of which general convulsions have occurred within a few weeks before or after the first appearance of laryngismus stridulus. The attack itself may end in a general convulsion, but this may be a result rather of the extreme cyanosis than of an extension of the convulsive phenomena.

Another indication of a convulsive tendency in rickety children, and one to the practical value of which I think too little attention has been paid, is the so-called "facial irritability." I mention it here because it is most often seen in association with laryngismus stridulus, but it occurs also apart from this, and occasionally even apart from rickets. The name is unsatisfactory, for the sign to which it refers is usually present in the limbs as well as in the face, and I have even found it in the limbs when it could not be obtained in the face; it would be better to speak of it as "nerve irritability." A gentle tap with the tip of the finger over the motor branches of a nerve produces a twitch in the corresponding muscle; perhaps the easiest place to demonstrate this is on the face, a tap on the facial nerve where it emerges on the face just in front of the ear produces a twitch of the orbicularis palpebrarum, and sometimes of several of the neighbouring muscles; and similarly a tap over the lower branches of the facial nerve just below and external to the angle of the mouth produces a twitch at the inner canthus of the eye, and sometimes of the muscles of the nose on that side. An exactly similar result can be obtained in the arm by tapping over the musculo-spiral nerve where it turns round the outer side of the humerus, or in the leg by tapping over the external popliteal nerve where it turns round the outer side of the fibula.

This nerve irritability is often very valuable as a danger signal. So long as a child shows this sign it is in danger of general convulsions; and I am inclined to go further, and say that the degree of risk can be judged to a certain extent from the degree of the nerve irritability; where many of the facial muscles are thrown into twitchings by each tap, or by mere stroking over the facial nerve, there would seem to be a greater liability to general convulsions than in those cases where the response is only slight or limited to a single muscle, usually the orbicularis palpebrarum.

Not only is this sign thus a useful indication of danger, and of the need for prophylaxis, but it may also guide us to some extent in the course of treatment, for it is often very noticeable that the diminution and cessation, under the influence of

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bromides, of convulsions which are dependent on rickets occur *pari passu* with diminution and disappearance of nerve or facial irritability. The effect of the drug on this symptom may indeed be taken as the outward and visible sign of the effectiveness of our treatment on the tendency to general convulsions, and may in this way serve as a gauge of the necessity for continuing or increasing our anti-convulsive measures. This nerve irritability is rarely found apart from rickets, and is usually associated also with laryngismus stridulus, and with another convulsive disorder which is also localised in its distribution, namely, tetany; it is, however, occasionally seen in infants who show a tendency to general convulsions during the first two or three months of life, when rickets is rare, and I have seen it in later childhood with epilepsy.

The relation of tetany to general convulsions is very close; in a considerable proportion of the cases it is preceded or followed by convulsions, which may also supervene at any time whilst the tetany spasm is still present in the limbs. I need not describe the ordinary tetany of infants, but I want to draw your attention to what has been called Trousseau's sign; for it is another indication of convulsive tendency, and, like the facial irritability, is sometimes of value as a danger signal. I will illustrate my meaning by an actual case:—A rickety infant, æt. 19 months, had laryngismus stridulus nine weeks ago; three weeks ago the infant had general convulsions; it has now very marked facial irritability, and although there is no apparent trace of tetany in the hands or feet, the effect of compressing the arm by encircling it just below the insertion of the deltoid muscle with one's forefinger and thumb, so as to compress the brachial vessels and nerves, is to produce in about thirty seconds the characteristic tonic spasm of tetany in the hand. This artificial method of exciting the tetany spasm is known as "Trousseau's sign;" and, as in this case, the presence of this sign is almost always associated with those other convulsive manifestations to which I have already referred, and is therefore an indication of danger.

And this brings me to the question of prognosis. What is the immediate risk entailed by an infantile convulsion, and what are the more remote risks?

In estimating the danger to life the first point to be taken into account is the age of the infant; there can be no doubt that the danger is much greater during the first year of life than subsequently. It has been calculated that 89·5 per cent. of the deaths from convulsions occur in infants under a year; and this high proportion is not, I think, altogether due to the fact that convulsions are commoner at this period than in older children, for it seems probable that there is no such disproportion between the frequency of convulsions in the first year and in the second as to account for this percentage of mortality in the earlier period. The following statistics are quoted by Lewis (Keating's Encyclopæd.) from Jamieson, who found that out of 365 fatal cases of convulsions in children under five years of age, 236 occurred within the first twelve months, and out of this number 165 occurred under the age of six months.

Age.	Number of deaths.
0—1 year	236
1—2 years	73
2—3 „	32
3—4 „	13
4—5 „	11

On the whole, it would seem also that the earlier the date of convulsions in the first year the greater the danger to life.

In considering the immediate prognosis naturally the severity of the convulsion, and its duration, must be taken into account; such a condition as I have already mentioned as comparable to the status epilepticus of adults is fraught with much more serious risk to life than the slight twitching with little or no loss of consciousness which occurs in many infants. At the same time one must always remember that even when they occur within a few days after birth, convulsions of extreme severity may end in perfect recovery, and that a fatal result is very exceptional. As long ago as 1789 it was pointed out by Underwood* that "children are much oftener supposed to die of convulsions than they really do, for though a convulsion frequently closes the scene, it has generally arisen from the great irritability of their nerves, and violence of the disease under which they have laboured."

But apart from the risk to life there are other very serious risks from infantile convulsions which must be considered. There can, I think, be no doubt that in a certain number of cases, probably

* 'Treatise on Diseases of Children,' p. 82, London, 1789.

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an extremely small proportion of the whole number of infants who have convulsions, a severe attack of convulsion results in permanent impairment of intellect. Take, for instance, such a case as the following:—A boy æt. 5½ years had been perfectly healthy, apparently, up to the age of six months, then he had thirty convulsions in two days; since then he has only had convulsions on two occasions, once at the age of twelve months, and again at three and a half years; but he is a boisterous, noisy idiot, he only learnt to walk at four years, and is dirty in his habits. His head is of normal size, his palate is high. Labour was natural and easy, and there was no difficulty in establishing respiration; there is, in fact, no apparent cause for idiocy apart from the severe convulsions.

It is, of course, extremely difficult to prove a causal relation in any such case, but it seemed even more apparent in the following:—A girl æt. 8½ years had learnt to walk and talk at the usual age, and seemed perfectly healthy up to the age of two years and eleven months; then she had ten convulsions in one day, and subsequently often eight or nine a day. Since the onset of these attacks she has ceased to talk, and has become imbecile; her tongue lolls out, and she takes little notice of her surroundings.

Happily such a result would seem to be extremely rare, but when it occurs it is probably more often from convulsions in very early infancy than from those occurring later. It is to be remembered, however, that while infantile convulsions are probably only a rare *cause* of idiocy, they are particularly common in association therewith. Statistics from 100 consecutive cases of idiocy or imbecility under my own care showed that infantile convulsions occurred in at least 22 per cent. of idiots; and if one included convulsions occurring after the age of two years (the end of infancy) the proportion would be 28 per cent., but out of these 100 cases there were probably not more than three in which there was any likelihood that the convulsions were the cause of the idiocy. Whilst, therefore, the risk of idiocy resulting from convulsions would appear to be small, we must remember that convulsions, if not the cause, may, at any rate, be the first indication of anything abnormal in an infant who is subsequently found to be an idiot.

But apart from idiocy there are other abnormal mental conditions which are probably in some cases the result of infantile convulsions. I have already mentioned that neuroses in older children may be a later manifestation of that same nervous instability which allowed the occurrence of convulsions in infancy; but it seems almost certain that occasionally infantile convulsions are the cause of mental abnormalities with which there is little or no impairment of intellectual power. Some of these infants who have had a severe attack of convulsions grow up eccentric even in their childhood, odd children with curious habits, hardly imbecile in the slightest degree, and yet "not like other children." Then, again, it seems almost certain, as I have pointed out elsewhere,* that a severe bout of convulsions in infancy may cause an arrest of moral development, with the result that the child from its earliest years is passionate, spiteful, or perhaps an incorrigible liar or thief.

Fortunately all these mental disturbances are rare in comparison with the frequency of convulsions, and, so far as I have seen, they would seem to result almost exclusively from very severe or prolonged attacks of convulsion; but they are worth remembering in all cases, and must "give us pause" when we are asked, as I was asked only a few days ago in such a case, Will the child's mind be affected?

It is not only the higher functions of the brain, however, which may suffer permanent damage, but also the motor functions. Some of the cerebral palsies of children, particularly the so-called "infantile hemiplegia," may be the direct result of a severe convulsion. Whether the intense venous congestion during the attack causes some hæmorrhage on the surface or into the substance of the brain, or whether this venous congestion so profoundly modifies the cells of the cortex that their function is henceforth impaired, may be uncertain, but of the clinical sequence there can be no doubt. Here again, however, prognosis must be wary; there are cases in which a complete hemiplegia, differing in no way from the initial stage of an ordinary permanent infantile hemiplegia, results from a severe infantile convulsion, and, after lasting for several hours, gradually disappears completely. In one infant æt. 19 months,

* Goulstonian Lectures, 1902.

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who was under my care at King's College Hospital, the left side was paralysed for four hours after a convulsion. In another, paralysis also of the left side remained for three hours only. In both these cases the paralytic affection was merely transitory, but had they been seen during the paralysis there would have been nothing to distinguish them from cases of infantile hemiplegia beginning with a convulsion; and I would remind you that fully 50 per cent. of cases of permanent infantile hemiplegia begin with a convulsion, although perhaps only a very small proportion of these have been caused by the convulsion.

There are other results which, although less serious, have some practical importance. In the later months of infancy, when speech is in process of development, a convulsion may result in stuttering, and if we regard stuttering as a disturbance of co-ordination we might almost have expected that a convulsion might produce it. Squinting also is undoubtedly in some cases a direct sequel and result of such an attack, whether the squint be temporary, as it is in some cases, or permanent, as it is in others; and this fact of the occurrence of squint, apart from more serious results, is worthy of note, for the question of commencing meningitis is so often raised by convulsions, and may be extremely difficult to determine. Take for instance the following case:—An infant three months old had been restless and sleeping badly for five days, and then had eight convulsions within a few hours. Two days later the doctor noticed slight retraction of the head, and the infant became much flushed at times. The bowels were very costive; there was no vomiting. On the ninth day of the illness more convulsions occurred, the infant was apathetic, there was a squint, and the fontanelle was bulged. At this stage I saw the infant, and found it as described, drowsy, and with very marked bulging fontanelle, but with no definite squint, although this was said to have been present earlier in the day. There was no paralysis anywhere, but there was an occasional momentary twitch of the hand. There was no stiffness of the neck at that time; the abdomen was natural. On the whole the balance of evidence seemed to be against meningitis, and this opinion was confirmed by a steady convalescence under chloral and bromide.

Another point which must be considered in prognosis is the relation of infantile convulsions to later epilepsy. When a child of two and a half or three years is brought with a history of convulsions from early infancy it is no easy matter to say whether the present attacks are to be regarded as epileptic or not; and, indeed, who shall decide where the line is to be drawn? There are cases in which convulsive attacks, epilepsy if you will, occur at intervals from early infancy onwards to the end of childhood and into adult life; but, so far as my own experience goes, I should say that out of the enormous number of infants who have convulsions an extremely small proportion become epileptic, at any rate before the age of puberty. This, be it observed, does not necessarily contravene the fact that of cases of epilepsy in later life a considerable proportion, about 12 per cent. according to Gowers, or 40 per cent. according to Osler, have begun during the first three years of life as infantile convulsions.

Even when infantile convulsions have persisted beyond the end of the second year they usually cease before the end of the fourth year, and often before the end of the third year.

But without any direct continuity it seems likely enough that, like the neuroses to which I have already referred, epilepsy also should be an occasional manifestation in later life of the nervous instability which conduced to convulsions in infancy.

With regard to the prognosis of the local convulsive conditions to which I have referred, you will gather from what has been already said that the chief risk both of laryngismus stridulus and of tetany lies in the tendency to general convulsions which these local affections indicate; but the former at any rate has also danger of its own, namely, the possibility of asphyxia during the closure of the glottis by the convulsive spasm; and although no doubt this is a more likely occurrence where the attacks are severe, I would strongly advise you never to say that a case with apparently mild laryngismus stridulus is free from risk, for at any moment a more severe attack may occur and end fatally. I have seen such an unexpected ending more than once, and have had occasion to regret the favourable opinion which I had formed. I remember in particular an infant of seven months who had what appeared to be quite a slight degree of laryngismus stridulus; it was well nourished, with only a moderate degree of rickets, and some facial irritability. It was attending here

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as an out-patient, and one day on its way home in the tram it was suddenly seized with an attack apparently of laryngismus stridulus; the parents rushed with it to a doctor's house which they were passing, but the child was dead by the time they reached there.

Tetany *per se* involves practically no risk to life, although in very rare instances affection of the respiratory muscles by the tetany spasm has ended fatally. But such affection of the respiratory muscles must, I think, be an extreme rarity; amongst a very considerable number of cases of tetany in infancy and early childhood which have come under my notice I have never seen such an occurrence.

In all these conditions, however, the risk of general convulsions is a very real one, and I come now to the consideration of their treatment. There are few of the exigencies of childhood which are more alarming and distressing to parents than convulsions in a child, and it is necessary in most cases to adopt some active treatment immediately, almost as much for the parent's sake as for the child's.

The traditional warm bath has, at any rate, the merit that it is almost always available; and if it does no good, it is at any rate not likely to do any harm. The hot water should be at a temperature of 95°—98° F., and the child should be kept in it, immersed to the neck, for about five minutes. In some cases this seems to have a quieting effect, and perhaps the benefit is increased by placing a cold or iced water compress over the child's head.

Far more effectual than the bath is the administration by rectum of bromide or of chloral; and here I would insist upon the greater effectiveness of chloral, and its value in any case of prolonged or frequently repeated convulsions. I have sometimes seen a small dose of chloral put a stop to the convulsions which had continued in spite of large doses of bromide.

But whether bromide or chloral is used, the drug will often require to be given with a free hand; and especially in dealing with the serious condition which I have compared with the status epilepticus of the adult, heroic doses may be necessary.

Even very young infants take chloral well in small doses. I saw with Dr. Key, of Southsea, an infant fifteen days old who had been in convulsions at short intervals for twelve hours. Potassium bromide had been given by the mouth in doses of $3\frac{1}{3}$ grains, which for the last three hours had been repeated hourly; some diminution of the convulsions had occurred, but there were still frequent twitchings of the limbs. Chloral gr. $\frac{1}{2}$ was then ordered immediately, and to be repeated alternating with the bromide every three hours; and no further convulsions occurred. By the rectum a grain dose of chloral might have been given to an infant at this early age, and if necessary might have been repeated in two hours' time; whilst for an infant three months old two grains might be given by rectum at intervals of three hours if necessary. At the age of twelve months two and a half grains may be given by the mouth, or double that dose by rectum if the case is urgent, but probably in most cases a dose of one grain by mouth repeated every three hours will be sufficient.

Morphia has been recommended, and I have seen it do good in these cases; you may give as much as a forty-eighth of a grain hypodermically to an infant six months old. I have, indeed, given more than this, but I would advise you rather to begin with less, and always feel your way carefully in the use of morphia hypodermically for infants; a sixtieth or even a hundredth of a grain may be quite sufficient. It is better to err on the side of safety.

Amyl nitrite has sometimes done good; it may be given by inhalation from capsules containing one minim.

If these measures fail the inhalation of chloroform is often of value, although prolonged administration may be necessary. Other remedies which have had their advocates are lancing of the gums where these are swollen and tense, the application of one or more leeches behind the ears, and the administration of oxygen.

More satisfactory than any remedial measures is the prophylaxis of convulsions. As I have already pointed out, there are several little indications, such as the presence of "facial irritability," or of those slight manifestations which the mother calls "the inward convulsions," which may apprise us of the tendency to the more serious general attack, and in such circumstances much may be done in the way of prevention.

Perhaps the commonest exciting cause of a convulsion is constipation; I select this out of many exciting causes which may call for treatment

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because I feel sure that, especially in the convulsions which occur during the first few months of infancy, constipation is so often the determining cause that regulation of the bowels should be our first thought in prophylactic measures. In rickety infants the administration of cod-liver oil has a very striking effect in reducing the nervous instability, and this effect is noticeable before there is any amelioration of the osseous changes. When there is marked facial irritability, or where convulsions have already occurred, I usually order one or two grains of bromide with an emulsion of cod-liver oil, and the rapid disappearance of the convulsive tendency therewith is often very noticeable.

With regard to the treatment of laryngismus stridulus, it has been held by some that the rational treatment of this disorder is to give "tone" to the nervous system by cold douches to the back, by fresh air, and by good hygiene in general, and that the administration of bromides is unnecessary. But whilst I would not for a moment dispute the value of such hygienic measures, I would remind you of the very real risk of sudden death during the convulsive spasm of the larynx, and on this ground I strongly advise you to give bromide in laryngismus stridulus, combining this drug with Ol. Morrhuæ, and, of course, not neglecting the hygienic treatment. There can, I think, be no doubt that the attacks are rapidly diminished, both in frequency and in severity, by the administration of bromides, or of such sedatives as chloral or phenazone, and in so risky a disease we cannot afford to adopt any line of treatment which means a slower, if not less sure, cessation of the attacks.

During the spasm of laryngismus stridulus a hot sponge applied over the larynx, or cold water splashed over the chest or face, may shorten the attack. In one fatal case, in which I happened to be at the bedside when the attack occurred, the spasm was so prolonged and the closure of glottis so complete, that I believe nothing short of tracheotomy could have saved life; but who would not hesitate—perchance, as in this case, hesitate a few seconds too long—to adopt such treatment for a condition which his experience tells him is usually quite transient? The treatment of tetany involves not only the prophylaxis of general convulsions on the lines which I have already indicated, but particularly careful attention to the state of the bowels; the very striking relation of tetany to a preceding diarrhœa or some abnormal character of the stools strongly suggests that the toxic influence, whatever it may be, which results in tetany, arises usually from the gastro-intestinal tract, and that our therapeutic measures must deal with this. As in laryngismus stridulus, the use of bromides is not only of value in relieving the local spasm, but has the advantage also of reducing the risk of supervention of general convulsions.

CERTAIN USES OF ELECTRICITY IN DISEASES OF THE NERVOUS SYSTEM.

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(*Continued from p.* 96.)

THE alterations in the electrical reactions met with in every important disease are fully described below.

1. DISEASES OF THE UPPER MOTOR SEGMENT.—As already stated, in diseases of the upper motor segment there are no electrical changes, or at the most some slight diminution to both currents in those muscles which are wasted and diseased.

2. DISEASES OF THE SPINAL CORD.—*Progressive Muscular Atrophy* (by this term I mean a disease due to chronic degeneration of the cells, chiefly in the anterior portion of the grey matter).—The electrical reactions in this disease are very interesting. At first there will be simply diminution to both faradic and galvanic currents in the wasted muscles. The nerves may show similar changes. These may be the only alterations in the electrical reactions for some time. At any time during the disease one may obtain partial R.D., but usually when it is well advanced. There is another important point to remember: sometimes one part of a muscle may respond fairly well to electrical stimulation; the remainder may show partial R.D., or hardly respond at all to either current. In late stages we may obtain complete R.D. if the atrophy is advanced, but partial R.D. is far more common here, as in all other palsies. Lastly, some muscles may fail to respond to either current. The electrical reactions in this disease are often very hard to obtain, as the wasting takes place in such a peculiar manner, many sound fibres lying side by side with degenerated fibres in the same muscle.

In looking through the records of the Electrical Department of St. Thomas's Hospital, during the last two years, of the cases seen of progressive muscular atrophy, there were only three which showed complete R.D., as defined by Erb, in any

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of the affected muscles, but partial R.D. of every variety was very common. Erb,* on the other hand, states, "I am obliged to regard this R.D. as a regular occurrence in typical progressive muscular atrophy."

Sir William Gowers† mentions some cases of this disease which have a rapid onset, or which advance rapidly some time during the progress of the disease. The paralysis is followed by rapid wasting. In such cases, this same authority states that there may be quick extinction of the faradic reponse, with actual exaltation of the galvanic, and that the reaction of degeneration may be present with all its characteristics.

Acute Anterior Poliomyelitis.—In this disease there is complete or partial R.D., and the electrical reactions are of great importance both for prognosis and diagnosis.

It will be found at the end of the first week in severe cases, on examination of the paralysed limb, that certain muscle groups have completely lost their faradic irritability, and that they may rapidly show changes with the galvanic current characteristic of complete or partial R.D. Other muscle groups remain intact to the faradic current, or only show some slight diminution. These muscles usually recover their motility rapidly, whilst the muscles which show advanced electrical changes will probably, *but not necessarily*, be permanently paralysed and wasted. The galvanic irritability usually slowly falls as the muscle-fibres degenerate, and completely disappears in cases which have not been treated by massage or electricity in about eighteen months. On the other hand, even those muscles which show well-marked electrical changes early in the disease may recover to a certain extent. These facts are much easier to write than to obtain; we must always remember that children strongly dislike their electrical reactions being taken, and therefore move about as much as possible; their skin is generally much more resistent than in adults, and there is a large amount of adipose tissue, which considerably impedes the efforts of the observer to obtain muscular contractions, and also may disguise the wasted muscles, if only a superficial examination is made of the paralysed limb. Lastly, we must always remember, if asked to give a prognosis in these cases, that although the affected muscles may completely recover, yet the antagonistic muscles may produce severe deformities during the process of repair.

Subacute Anterior Poliomyelitis.—Partial or complete R.D. is obtained in the wasted muscles, but in these cases the power of the atrophied muscles may return before we obtain normal electrical reactions.

Chronic Myelitis.—The electrical reactions in those cases in which there is destruction of the cells of the anterior cornua, in the cervical or lumbar enlargement, will show the presence of either partial or complete R.D. in the atrophied muscles.

*Syringomyelia and Tabes Dorsalis.**—If accompanied by changes in the anterior horn cells, there will be complete or partial R.D. in the wasted muscles; in the former disease, of course, it is far more common.

3. Unclassified Diseases. — *Functional Disease.*—It is a well-known fact that many patients suffering from hysteria frequently show patchy or diffuse analgesia, or at any rate some dulness to painful stimuli. This analgesia is equally well illustrated by means of electricity. These patients often complain that they are unable to feel strong faradic currents, so strong that they would cause normal people intense pain. Again, patients suffering from hysterical monoplegia or hemiplegia will generally show a loss of diminution of electrical sensation in the affected limb or limbs, while over the rest of the body sensation will be found to be normal. Lastly, Professor Raymond† refers to the urethral anæsthesia which is present in some cases of functional disease. He was able to pass an electrode connected with one of the poles of the battery along the male urethra; the other pole was placed on the glans penis; a maximum faradic

* Professor Erb, von Ziemssen's 'Handbook of General Therapeutics,' 1887; English translation by A. de Watteville.

† Sir William Gowers, 'Diseases of the Spinal Cord and Peripheral Nerves,' 1899, vol. i, p. 539.

* I have noticed in these diseases, and also, but to a less extent, in hysteria, peripheral neuritis, etc., that when patients have electrical anæsthesia there is sometimes considerably diminished muscular contractions to both faradism and galvanism of the muscles in the anæsthetic area. Of course, I refer here to cases where the muscles themselves appear to be quite normal.

† Professor Raymond, "Anesthésie cutanée," 'Revue de médecine,' 1891, p. 395.

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current was passed without any effect. He refers to a similar anæsthesia of the rectum.

Arthritic Muscular Atrophy (as met with in rheumatoid arthritis, gonorrhœal arthritis, etc.).—Here we have a slight and equal diminution to both currents in the atrophied muscles, but never any very marked electrical changes. In cases in which the arthritic atrophy is associated with "lower segment paralysis" we shall obtain partial or complete R.D.

Landry's Paralysis.—In pure cases of this disease the electrical reactions are normal. In some of the reported cases of "Landry's paralysis" there has been partial or complete R.D., but it is probable that they were examples of lesions of the peripheral nerves, cord, or both.

Tetany.—The electrical reactions here form perhaps the most characteristic feature. They are of great value, as they are present both during the attacks and in the interval.

All nerves may be found to have greatly increased excitability to both the faradic and galvanic currents, especially the facial nerve. We must, however, always remember that increased nerve excitability is a most misleading statement, as patients vary so much in regard to this phenomenon, even those who show no other abnormal features. The nerves also respond briskly to a small strength of current, especially galvanism, but also with faradism.

Galvanic Reaction of the Muscles.—The muscles more readily respond with the positive pole, either on closure or on opening of the current, than with the negative pole. The contraction is not a short, sharp contraction as in health, but is prolonged, so that tetanus results. Thus the formula will be ACC > AOC > KCC, or AOC > ACC > KCC. The anodal opening contraction, in the form of a tetanic contraction, is the most characteristic feature of this disease.

At the present time a girl suffering from chronic tetany is attending the electrical department of St. Thomas's Hospital with these reactions, except that they are *not* present in the interval of the attacks.

Myasthenia Gravis.—Exhaustion of the muscles stimulated with the faradic current is obtained in this disease. If you excite, *e. g.*, the arm muscles with the faradic current for about a minute or more, they become exhausted and cease to respond; and if the stimulation is continued after exhaustion, it will be some time before the muscles recover, according to the length of time stimulation is allowed. This reaction is not obtained with the galvanic current.

Thomsen's Disease, or Congenital Myotonia.—Professor Erb,* who was the first to describe the electrical reaction in this disease, applied the name "myotonic reaction." The motor nerves act on the whole in a normal quantitative and qualitative manner to both currents. All single shocks give short contractions, but on continued excitation with a strong faradic current or labile galvanic current (*i. e.* the negative and active electrode is made thoroughly wet, and then rapidly and energetically applied with a rubbing movement in the course of the nerve-trunk or muscle to be excited, and the current is employed of sufficient strength to cause a vigorous and wavy contraction) a very characteristic persistence of the contraction occurs after the cessation of the irritation. The muscles very easily respond to the faradic current, and with currents of moderate strength always show persistence of the contraction.

Galvanic excitation of the muscles causes a slight increase of excitability; with strong currents the contractions are slow and tonic, but only occur on closure of the current. A stabile galvanic current is obtained by holding the negative and active electrode quite still. It is best seen by placing the electrode close to the tendon of the muscle which is to be examined; it will cause rhythmical wave-like contractions, starting from the cathode and spreading to the anode. These reactions have not always been found, but most German physicians adhere to Erb's views. Jolly, quoted by Sir William Gowers,† has found that repeated stimulation by either current, without too long an interval between the successive stimulations, tends to render the after contraction less and less evident, until it ceases to occur.

Pseudo-hypertrophic Paralysis. Simple Idiopathic Muscular Atrophy.—In the former disease there is equal diminution to both currents in those muscles which are wasted, or in which there is marked weakness. The electrical reactions in the latter disease are similar, except that there is often much more diminution to faradism and galvanism than

* Loc. cit.

† Sir William Gowers, loc. cit., p. 603.

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can be accounted for by wasting and weakness in the affected muscles. There is *never* any trace of the reaction of degeneration in either disease.

In muscular atrophy from disuse, in which there is no neuritic change, the electrical reactions are normal, or at most there is some diminution to both currents. Partial or complete R.D. is never present.

Family Periodic Paralysis.—The electrical reactions which are present in this rare disease I shall quote from Dr. Singer's * paper on the subject. He says, "In the intervals between the attacks the muscles all react normally. During an attack there is a gradual reduction of excitability, both to faradism and galvanism *pari passu* with the loss of voluntary power, until with complete palsy there is complete loss of excitability to both currents. With the reduction there has never been any change either in quality of contraction, nor has there been any polar change to galvanic stimulation. As voluntary power recovers there is a gradual return of excitability to both currents. The facial muscles have always reacted normally when stimulated through the nerve or directly, even at the height of an attack."

Family Form of Muscular Atrophy in Children, with Spinal Lesion.—In those muscles which are atrophied the reaction of degeneration has been found.

Myxœdema.—The electrical resistance of the skin has been found to be increased.

Exophthalmic Goitre.—In this disease exactly opposite results are obtained to those in myxœdema. Charcot and Vigouroux were the first to notice that there was a decrease of the electrical resistance of the skin. In health the average resistance of the skin to galvanism is 4000 to 5000 ohms. In eight cases in which Wolfenden † investigated this point in exophthalmic goitre the skin only gave a resistance varying from 500 to 700 ohms, and in two of the cases it varied between 200 and 300 ohms. This phenomenon is usually considered to be due to the excessive moisture of the skin which is present in so many cases, owing to the dilatation of the surface capillaries, and to the activity of the sweat-glands; but it cannot be entirely due to these facts, for this decrease of the electrical resistance of the skin is not present in every case of Graves's disease. At any rate, this sign is by no means pathognomonic.

Lastly, there are many cases which appear in the electrical department of a large hospital which follow no well-defined classification, and in which there are electrical reactions characteristic of no special disease.

4. Diseases of the Muscular System.—*Volkmann's Contracture.**—In pure cases of this disease the electrical reactions are normal. This fact is most important in the diagnosis of the deformity.

Acute Myosotis.—There are no electrical changes unless atrophy follows. In such a case there will be a diminution of the electrical reactions to correspond to the muscular atrophy.

5. Diseases of the Peripheral Nerves.—*Peroneal Form of Progressive Muscular Atrophy.*—The electrical reactions are of very great value in the diagnosis of this disease. Partial or complete R.D. may be present in the atrophied muscles. In those muscles which are not atrophied, and in which there is a normal amount of power, there may be great difficulty to excite contraction by means of electricity. Professor Oppenheim † describes a case with severe disturbances of the electrical excitability over the whole body, but the only atrophied muscles were in the lower extremities.

In other diseases, also of the lower motor segment, electrical changes may be met with in muscles which are not atrophied, and in which the power seems to be normal.

Paralysis of the Motor Portion of the Trigeminal Nerve, which supplies the muscles of mastication. If due to a peripheral lesion, or nuclear, partial or complete R.D. may be found.

Paralysis of the Seventh Nerve.—This has already been fully discussed, but two further cases are appended here to illustrate some of the important but difficult problems met with in this unpleasant deformity.

(1) A girl æt. 26. Rapid facial paralysis followed

* H. D. Singer, "Family Periodic Paralysis," 'Brain,' part xciv, summer, 1901.

† R. Norris Wolfenden, "A New Point in the Diagnosis of Graves' Disease," 'Practitioner,' 1887, vol. xxxix, pp. 422—429.

* L. S. Dudgeon, "Volkmann's Contracture," 'Lancet,' January 11th, 1902.

† Professor Oppenheim, 'Diseases of the Nervous System,' 1901 (American translation).

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an operation for mastoid disease in the summer of 1900. Three weeks after the operation there was no response to the faradic or galvanic current at any motor point for the facial nerve. Upper facial muscles did not react to the faradic or galvanic current. The lower muscles showed the characteristic complete R.D. The prognosis in such cases is generally bad. In this case it proved a not uncommon rule, for when she was seen in the electrical department in December, 1901, the facial paralysis was absolutely complete, with no electrical response of any sort in the facial nerve or muscles.

(2) A girl æt. 24. Right-sided facial paralysis rapidly following on mastoid operation. About three weeks after the operation, in December, 1901, she came to the electrical department, St. Thomas's Hospital. There was no response of the facial nerve or muscles with the faradic current. With the galvanic current the upper fibres responded by a "twitch;" the lower fibres by a slow, sluggish contraction, with, however, no polar change. She was ordered galvanism for ten minutes daily. The prognosis here was very difficult to give; it might improve during the next six or nine months, it might follow the example of the case quoted above.*

Alcoholic Neuritis.—Here we have rapid loss of faradic and galvanic irritability in the nerves of the affected part, followed by the changes characteristic of partial or complete R.D. in the affected muscles. It is often most characteristic that muscles which do not appear to be much wasted, present such marked changes as the partial or complete reaction of degeneration. In some cases muscles which show increased galvanic irritability, which is met with in complete and partial R.D., rapidly lose all irritability, owing to the severe degenerative changes in the muscle-fibres.

Sir William Gowers† describes an extremely rare alteration in the electrical reactions, *e. g.* a preservation of some galvanic irritability in the nerves as well as in the muscles, faradic irritability being lost in both. These changes are of the greatest importance, as in those cases in which the muscles fail to respond the prognosis is bad, for when improvement does take place it is always very slow. On the other hand, we must always remember that normal electrical reactions may only be obtained some time after power has returned.

In post-diphtheritic paralysis the electrical reactions will follow the same course as in any other degenerative lesion of peripheral nerves.

Leprous Neuritis.—It is stated by different authors on this subject that, as the lesion is generally so chronic, the electrical diminution is very slow, but in very marked and long-standing disease there may be no electrical response in the affected muscles. From observations I have made on many cases of this disease lately, it seems that the electrical reactions are sometimes similar to those of progressive muscle atrophy, *i. e.* portions of certain muscles are severely affected, other portions are normal.

Sciatica.—There is sometimes diminution of the electrical reactions in the nerve and corresponding muscles. In neuritis of this nerve partial or complete R.D. may be obtained.

*Simple Nerve Palsy.**—In complete division of a peripheral nerve, complete or partial R.D. is obtained.

In lesions of the nerve-roots, *e. g.* cervical pachymeningitis, complete or partial R.D. may be obtained in the atrophied muscles.

Arsenical Neuritis.—The electrical reactions are summarised by Kelynack† as follows:—"Qualitative and quantitative changes in the electrical reactions have been well marked, and the so-called reaction of degeneration has been observed in bad cases."

"Electrical testing has shown in convalescing cases an absence of the reaction of degeneration."

Obstetrical Paralysis.—In two cases of Mr. Robinson's‡ Dr. Turney found no alteration in

* This case had in September, 1902, almost completely recovered, but the electrical reactions were those of complete R.D.

† Sir William Gowers, loc. cit., p. 157.

* I found on one occasion, in a case of facial paralysis, in which there was no response either of the nerve or muscle to faradism or galvanism, that when the nerve was cut down upon so as to see if the cause of the paralysis could be determined, both the nerve and muscles responded to both currents on direct stimulation. This shows what an important part the epidermis plays in electrical examinations.

† T. N. Kelynack, 'Arsenical Poisoning in Beer-drinkers,' 1901, p. 38.

‡ H. B. Robinson, "Traumatic Birth Palsies of the Upper Extremity," 'St. Thomas's Hospital Reports,' 1897, vol. xxvi, p. 161.

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the electrical reactions. In two cases I have seen during the last eighteen months the electrical reactions were normal, and both cases completely recovered with massage, but with no electrical treatment. In the common variety of this paralysis, *i. e.* Erb's or the upper arm type, it is stated that in all severe cases the reaction of degeneration is obtained.

In the facial type, which recover in about two days to two weeks, the electrical reactions are normal and the prognosis good. In very severe cases complete or partial R.D. may be present. In these cases the prognosis is naturally worse. Gowers * states that most obstetrical cases slowly recover.

Lead Paralysis.—This is generally in the form of double wrist-drop. There may be anything from partial to complete R.D. in the muscles supplied by the post-interosseous branch of the musculo-spiral nerve. The irritability to galvanism is for a time increased, and there may be also qualitative changes. In those cases in which other muscles and nerves are also affected they will show similar changes. There is at the present time a patient suffering from chronic lead poisoning attending the electrical department, with considerable wasting of the thenar and hypothenar eminences, and the interossei as well as the extensor muscles of both forearms. All these extremely atrophied muscles show well-marked partial R.D., except the muscles of the thenar eminences of both hands, which do not react to either current. This man has been about fourteen years a lead worker, and has never received any treatment for his paralysis. In lead poisoning we also meet with cases where certain muscles show partial or complete R.D., but the motor function may be practically normal, and other muscles which are not wasted, but only weak, may present similar changes. The prognosis is of course much better in those cases in which there is a very slight alteration in the electrical reactions, but most of the acute cases with partial or complete R.D. generally recover under treatment, but often only to relapse when patients resume the same occupation.

Hypoglossal Paralysis.—This nerve may be injured during operations in the neck, and an opinion is sometimes required whether it is cut or otherwise. The testing electrode is placed on the motor point for the nerve in the neck, and a weak faradic current is used; the other electrode is placed in any convenient position on the body. The tongue is also examined locally with weak faradism and galvanism.

An old man, who had been operated on for a growth in the neck, was sent to the electrical department with unilateral atrophy of the tongue. It was found that the hypoglossal nerve reacted to faradism (four weeks after the operation), whilst the tongue muscles also reacted, although sluggishly, to both currents. In this case it was obvious that the nerve had not been cut. It is important that we should only apply a very weak current to the tongue, and we must remember that as the tip of the tongue in unilateral atrophy curls round, we must not mistake the contraction of the muscles of the sound side for the paralysed. Unilateral and bilateral atrophy of the tongue also occurs from nuclear lesions.

Sensory Phenomena-nerves of Special Sense.—These can usually be excited by galvanism.

Optic Nerves.—If the positive and inactive electrode is placed on the neck, and the active electrode very gently on the closed eyelid, flashes of light will be experienced at make and break. In cases of optic atrophy this phenomenon may be absent.

Olfactory Nerves.—Stimulation of these nerves may cause peculiar sensations of smell. This phenomenon may also be absent in certain diseases of the nervous system, *e. g.* tabes dorsalis.

Gustatory Nerves.—These are stimulated by placing the electrodes on either cheek or on the tongue (in the latter case, of course, only a very weak current can be used). On the side of the anode, a metallic, alkaline, and even, but rarely, acid sensation may be experienced; on the kathodic side it is usually saltish, *never* alkaline.

Auditory Nerves.—Certain sensory, visual, and gustatory phenomena may occur during the examination of this nerve, because it is so deeply situated, and therefore requires a strong current for excitation. The active electrode is placed in front of the auditory canal, firm pressure is employed, and the inactive electrode is held to the patient's back. A buzzing, whistling, or musical sound is produced with a strong current at KC, only a feeble sound will be heard at AC, while at AO and KO there is no auditory conduction.

Numerous abnormal changes may be found on examination of patients by this method, suffering from ear diseases, *e. g.* hyperæsthesia, diminution, and anæsthesia, etc. The last mentioned only occurs in severe lesions. Lastly, severe chronic diseases of the internal and middle ear may produce polar and quantitative changes.

(*To be concluded.*)

* Sir William Gowers, loc. cit., p. 113.

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A CLINICAL LECTURE

ON

THE SPECIAL CAUSES OF DEATH DURING AND AFTER OPERATIONS ON THE MOUTH AND THROAT, AND THE BEST MEANS OF AVERTING THEM

(*Preliminary Laryngotomy*).

Delivered at St. Bartholomew's Hospital, May 28th, 1902.

By H. T. BUTLIN, F.R.C.S., D.C.L.

GENTLEMEN,—If you are largely engaged in surgery you will be frequently asked by anxious friends and parents whether the operation you recommend is quite without danger; and you will reply that, while no operation is without danger, this particular operation is either so little dangerous that the danger need not be seriously taken into account, or that the danger of the operation is fully justified by the nature of the disease which is to be dealt with. This is particularly the case in many operations which fall to my lot, in which the operation is performed for malignant disease in the mouth or throat. The risk is quite justified by the hope of curing or relieving the patient of a terrible disease, resulting in a terrible death. The risks of removal of the whole of the tongue and floor of the mouth, the upper and the lower jaw, a large part or the whole of the larynx, for example, are very considerable, and the mortality of some of these operations has not been lessened in the same proportion as the mortality of operations generally. Thus the mortality of removal of the upper jaw remains at about 30 per cent., much what it was thirty years ago; that is, a mortality of about one in three, an enormous loss of life when the general improvement in surgery is remembered.

In addition to the ordinary dangers attendant on every operation, these operations on the mouth and throat are attended by certain special dangers,

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and I am going to devote my lecture to-day to those special dangers and to the best means of meeting and averting them. I am not going to speak of the smaller operations, such as the removal of tonsils and adenoids, for these operations are performed by everybody everywhere, and each operator has his favourite method. I shall speak only of the larger and more serious operations, such as those to which I have alluded. And in order to impress on you more forcibly, or perhaps more picturesquely, their special dangers, instead of merely enumerating them, I will illustrate each danger by a case in which I have borne a part.

Sepsis.

Some time ago I was consulted by a gentleman who was suffering from a foul ulcerated cancer of the palate and tonsils, with glands in the neck which were almost certainly beyond the reach of operation. He suffered so much from the primary disease that I removed it very freely, and the question of removal of the glands was left for the moment. During the first three days he did so well that there seemed no doubt of his recovery; then his temperature rapidly rose to 103° or 104°. He became jaundiced and very ill, and in the course of three or four days he died with the symptoms of sepsis, but without any cough or sign of affection of any special organ. His case may be regarded as a pure case of sepsis, and perhaps it is fair to attribute the poisoning partly, not only to the impossibility of maintaining such a wound aseptic, but to the weak condition of the patient, which rendered him peculiarly susceptible to infection, and also to the fact that the disease which was removed was horribly septic, and may have directly poisoned the fresh wound of the operation.

Septic Affections of the Lungs.

Many years ago, after a severe operation on the larynx, the feeding was carried on through an œsophageal tube. It was one of my early cases. The tube was very large, and not, as I now think, well adapted for the purpose; it was passed with difficulty, and, as it was withdrawn, some of the contents passed into the larynx and down the trachea. This was followed by a rapid rise of temperature, and by the development of pneumonia, of which the patient speedily died. He died of septic pneumonia, almost certainly due to the decomposition of the liquid food which passed into the air-passages. The Germans have given this form of pneumonia the name of "Schluck-pneumonie," which may be translated swallowing-, or more justly, sucking-pneumonia, for food and foul discharges are often sucked down into the air-passages during inspiration. It commonly results in septic abscess or gangrene of the affected lung, and few people who are stricken with it recover. Let me give you another example of it to show how it may be due to another cause than food.

Some six or seven years ago a gentleman came with a doctor to consult me one morning. He had epithelioma of the side of the tongue, extending into the floor of the mouth, and attached to the lower jaw, but not very far back, and quite within the reach of an extensive operation. I knew nothing of this gentleman, nor did I know the doctor, and it was necessary to explain to one or both of them the serious nature of the malady, and how it might be removed by an operation, but not without considerable risk of life. You may imagine how distressed I was to find that both the patient and the doctor were, at that time in the morning, drunk; a circumstance not merely disgusting in itself, but which told a story of ill-regulated life not likely to fit a man to undergo the strain of a serious operation. The patient, with the happy-go-lucky assurance of a drunken man, was quite ready to undergo any operation, even to the "cutting off of his head;" but I would not hear of an operation until I had learned more about him, and had had a consultation on his case with my colleague, Sir Thomas Smith. The operation itself passed off very well until, when it was nearly finished, there came a gush of blood into the mouth, and the patient struggled, as people often do in the course of such operations, and then drew in a deep breath, and with that sucked blood down into his trachea. A sponge was thrust into the wound to stay the hæmorrhage, and the patient was turned far over on his side with the head down to let the blood run out of the air-passages, and so we cleared the left bronchus, but not the right. However, during the first three days the progress of the case was excellent, and I began to hope we had been frightened to little purpose; but in the evening of the fourth day his temperature rose, and he began to cough. The

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temperature scarcely fell again, and the cough increased, while the respirations became more rapid. With this the breath was offensive, and particularly so was the stuff which he spat up. Dr. Hall examined his chest, and found that, while the left lung was free, but little air entered the right lung, and that the signs of inflammation were on that side. He was quite of my opinion that the pneumonia was due to the clotting and decomposition of *blood* which had been sucked down into the right bronchus, and he gave a very grave prognosis. Twelve days after the operation the temperature rose to 106·2°, the pulse and respirations became much more rapid, and three weeks after the operation the patient died with all the symptoms of gangrenous abscess in the right lung.

Anæsthesia.

Now take a danger of another kind. A good many years ago, when Mr. Mills was the anæsthetist to this hospital—and he was an excellent anæsthetist—he was giving chloroform in a nursing home to a strong active man, about forty years of age, on whom I was about to operate for quite a small epithelioma of one tonsil. The gentleman was a man of substance, so his doctor came up from the country to be present, and I had my assistant as well. In addition, there were two nurses, so there were at least six persons in the room to look after the welfare of the patient. I was standing close by the operating table, waiting to put the gag into the mouth, when suddenly the patient struggled, and I saw a horrible change pass over his countenance, which became deadly pale, then hideously livid. He gave a kind of shudder, and I felt his pulse, only to find that there was no pulse, although he continued to breathe. Mr. Mills saw these changes exactly at the same moment that I did, and I feel sure that not ten seconds elapsed before we had the man's head over the end of the table and the tongue drawn out, and we were performing artificial respiration and striving to stimulate the heart to act again. All to no purpose, for the man was dead, not during the course of an operation, but before even the gag had been placed in his mouth. That is an illustration of danger due to anæsthetic; and, if you are disposed to think that there is no special reason for associating the kind of operation with this special danger, inasmuch as neither the disease nor the operation had anything to do with the death, which was purely due to the chloroform, I am quite ready to admit it. But I chose the case to illustrate the sudden faintness which may occur in the course of these operations, and which is sometimes distinctly occasioned by the anæsthetic, and which is overcome by such measures as we employed, but which would add enormously to the danger of such a condition as I am now about to illustrate by my first case of excision of the tongue.

Hæmorrhage.

It was on a winter's day many years ago. Almost the whole tongue was infiltrated with cancer and was bound down to the floor of the mouth, so that it could not be protruded, and the man was a drunkard, so the case was a bad one. Yet I am sure I approached the operation with confidence, begotten partly of youth and partly of a great lack of experience; and I thought I should relieve the patient if I did not cure him; and this I did in a manner which neither of us looked for, and much more quickly than he or I anticipated. I had the advantage of the advice and actual assistance of my colleague, Mr. Morrant Baker, who at that time had deservedly one of the best reputations in this city for operations for cancer of the tongue. But all was in vain. Scarcely was the patient under the influence of chloroform than I drew the tongue forward with threads, and taking a pair of scissors, cut underneath it to separate it from the floor of the mouth. But I cut too quickly and too far from the jaw, and suddenly there was a great gush of blood in the mouth. The patient was lying on his back, and the blood ran into the back of his mouth; he gave a great struggle, and with that gulped a quantity of blood down into his air-passages. His respiration ceased, and, do what we could to restore him, in a moment he was dead. And there, by the table, stood I, looking with dismay at the dead man, wondering not only how the accident had happened, but whether in the future I had not better leave such operations to surgeons more skilful than myself. My colleague, the kindest hearted of men, was good enough to assure me that the case was as hopeless a case of cancer of the tongue as he had seen, and that I had probably done the fellow a service by the abrupt termination of his disease.

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But he would be a poor creature and a bad surgeon who would be content with such a view as this. And, as the Department for Diseases of the Throat and a kind of chance gradually drove me to perform operations in the mouth and throat at frequent intervals, my constant endeavour has been to be able to perform them with greater safety to the patient and with a prospect of greater permanent success. You may say this death was due to hæmorrhage, but that would be only to a certain extent true. It was the combination of hæmorrhage and anæsthesia which was the actual cause of death, and it is these combinations of causes which are responsible for many of the fatal results of operations, far more frequently than one single cause.

Of course, it is possible for a patient to die of loss of blood before a large operation, on the upper jaw, for instance, is completed; or the loss of blood may have been so great that it is not recovered from, and the patient is said to have died of shock or of exhaustion. But such deaths ought to occur but rarely, unless in the old or very feeble.

Precautions against Sepsis.—The special dangers of these operations may, then, be summed up under the heads of sepsis, septic affections of the lungs, anæsthesia, and hæmorrhage. And these dangers are due to the seat and nature of the disease; to the entrance of substances such as blood, food, and discharges into the air-passages, where they decompose; to the combination of anæsthesia with hæmorrhage.

Against sepsis it is scarcely necessary to observe that although such operations cannot be truly aseptic or antiseptic, yet every precaution should be taken not to increase the danger at the time of the operation by lack of cleanliness of the hands and of the instruments which are used, of the sponges, of the ligatures, and sutures. And after the operation you will find, in addition to the frequent cleansing of the wound and of the cavity of the mouth with boracic lotion, or sanitas, or Condy's fluid and water, that iodoform in powder is one of the best local applications; it should be puffed on the raw surface three or four times a day, and may be used pure. Of preparation of the inside of the mouth, I have done but little, except by the removal of very bad teeth and stumps, for the means are very inadequate to the end. Nor have I used more than a few times preliminary injections of antistreptococcus serum, which was, I think, recommended for the purpose by Mr. Watson Cheyne. I had intended to use it in many cases, but deaths from pure sepsis are, on the whole, rare, and I think that there are better means for preventing septic affection of the lungs, which forms the gravest danger.

Precautions against Septic Affections of the Lungs.—To guard against the entrance and sucking in of gases and foul discharges, let me recommend you to place the patient generally on the side from which the disease has been removed, and only to allow one flat pillow, so that the head lies very low, and even to encourage that it shall hang over the edge of the bed from time to time, that the discharges may tend to run out of the mouth instead of into the back of the throat, where they are liable to collect, and are often swallowed in considerable quantity, as the patient may be unable or unwilling to take the trouble to spit them out.

During the first day or two days let the nourishment be given chiefly or entirely by the bowel; and in severe cases where there is doubt whether feeding by the mouth may at first be very difficult, and whether some of the liquid may make its way down the air-passages, be sure to superintend the first feedings yourself, and use only water (which has been sterilised by boiling) for the first experiments. For, if it should by chance run into the trachea, it is not so dangerous as milk or beef-tea. If the patient must be fed by means of a funnel and tube, there are several details of great importance. A bulb-pointed catheter about No. 7 or 8 English should be used. To the upper end of it light and small india-rubber tubing is fixed, and into the upper end of the tubing the nozzle of a small glass funnel. The catheter is dipped in glycerine, and is passed halfway down the œsophagus, not farther. The funnel is then filled, and during the filling the india-rubber tube is pinched, so that not a drop runs down the catheter. By straightening the tubing some of the air which it contained is coaxed to rise to the top of the liquid in the funnel. But it is of small consequence whether a little air is carried into the stomach. The liquid is allowed to run very slowly at first, and if the patient coughs or retches ever so little the tube is at once tightly pinched, or the funnel

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is lowered below the level of the mouth to stop the flow. Otherwise some of the contents of the stomach may be brought up into the mouth and sucked down into the trachea. When the food has been administered, the funnel is raised as high as it will go to empty the funnel and tubing, and the catheter is quickly withdrawn; and while this is done the tubing is pinched so tightly that not a drop escapes into the mouth. These details seem to be almost trivial, but they are of vital importance, and the neglect of any one of them may result in a catastrophe.

A good nurse and an intelligent patient will carry out these instructions after two or three demonstrations with perfect ease and safety, and the patient will regulate the rapidity of the flow of fluid according to his liking and convenience. Three feeds a day, each of a pint, suffice for most patients, and are more by half a pint than some patients can digest after operation.

Precautions against Hæmorrhage.—The best method of dealing with hæmorrhage and the administration of the anæsthetic during the operation was a subject of consideration and experiment during several years. For operations on the tongue, the écraseur was the instrument of predilection during my early years at the hospital. In spite of the improvements which Mr. Morrant Baker introduced into its use, it gave way gradually in favour of Whitehead's operation with scissors, and gradually died out, so that I suppose nobody here has ever seen it used. When we grew accustomed to Whitehead's method, we found it a great improvement on all previous methods, and admirably adapted to simple cases of excision of the whole or half of the tongue. But in the larger and more complicated cases there is more hæmorrhage, and greater fear of the sucking down of blood than is desirable. Whitehead placed his patients almost in the sitting posture, while (the German surgeon) Rose's posture, in which the patient is laid upon his back, with the head hanging down over the end of the table, was favoured by some surgeons. I liked neither the one nor the other of these postures. The sitting posture is of more advantage to the surgeon than to the patient, and does not meet the difficulty of blood collecting in the mouth and at the back of the throat. And Rose's posture I have always strongly discountenanced. You have only to place yourself upon a flat bed or couch upon your back, and hang your head down over the end of the couch, to understand why. The blood "runs to your head," to use the common expression, and the veins are full to overflowing. I cannot understand how any surgeon can undertake a large operation on the mouth or throat with the patient in such a posture. After trying the *lateral* posture for adenoids, and growing accustomed to the difference in the bearing of the parts, I began to use it for the larger operations, and found it a great improvement on every other I had tried. The head is placed on a flat pillow, and the patient is so far turned over on his side that the back of the head is a little higher than the front, and the blood collects in the hollow of the cheek or runs directly out of the mouth. The disadvantages are that the operator must sometimes sit on a low chair in order to be on a level with the patient's mouth; and it is not always easy to maintain the patient in the same posture during a long operation. Still, the lateral posture was a distinct advance.

Preliminary Ligature of Vessels.—The preliminary ligature of vessels is only serviceable in certain cases. And even when the operation is on the tongue, I have never cared to perform it unless the neck had to be opened for the removal of lymphatic glands, or for some other reason. The ligature of the lingual artery is itself a long and troublesome operation in a thick-necked man on a foggy morning in the winter. And the fear always exists lest the vessels which were not ligatured in the mouth because they did not bleed, may not do so a few days after the operation. If, however, the neck is opened for the removal of lymphatic glands or for the better removal of deep-seated disease in the floor of the mouth, or of a portion of the lower jaw, the lingual artery may with advantage be ligatured just above its origin, or the external carotid may be tied.

Preliminary Tracheotomy.—It is not unnatural that the question of a preliminary tracheotomy should have presented itself to surgeons engaged in operations on the mouth and throat. It has been recommended several times within my recollection by writers in the medical journals; and perhaps by no one with greater force than by Mr. Arthur Barker a good many years ago. But it has never been generally popular among surgeons. The operation itself is often tedious, and there is

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frequently loss of more blood than is desirable, while the wound may be as troublesome to deal with as the wound of the principal operation. For several days after tracheotomy there may be difficulty in coughing up discharges and expectoration, which collect at the back of the throat. (In spite of its disadvantages, tracheotomy with the use of Hahn's tube is a wonderful preliminary to operations in the larynx or immediately above the larynx.)

Preliminary Laryngotomy. — For operations higher up, for removal of part or the whole of the tongue, for operations in the tonsillar region, the palate, the naso-pharynx, the inside of the cheeks, and on the jaws, I am about to recommend you to employ the preliminary operation, to which I have given a prolonged and careful trial during the last two or more years, and which many of you have seen me perform on Tuesday afternoons. At a meeting of the Laryngological Society towards the end of the year 1899, a boy was exhibited who was suffering from a very large and formidable tumour of the naso-pharynx and back of the nose, and our opinion was asked on the nature of the tumour and on the best means of attacking it by operation. Several of us spoke and gave advice according to our lights and knowledge, one of us that the palate should be split, another that the operation should be performed with the head hanging back, a third that there should be a preliminary tracheotomy, and so on. But Dr. James Bond, of the Hospital for Diseases of the Throat, said, "No, don't perform a tracheotomy, but rather a laryngotomy. The operation is trivial and quickly performed, and the tube is taken out immediately after the major operation. And during the operation put a sponge at the back of the throat." I was at once struck with the value of this suggestion, and how it could be applied to all operations on the mouth and neighbouring structures, and I determined that I would put it in practice at the first opportunity, and, if I was satisfied with it, would practise it again and again in order to ascertain its limits, its difficulties, and the dangers attendant on it. I have carried out this intention, and have performed the operation very many times, probably between fifty and sixty times at least, and I think I have learnt nearly all that is important; and I am about to impart this knowledge to you in order to spare you the trouble and disappointments which you might encounter if each one of you had to work out the details for himself. For if a preliminary operation is to be really serviceable, it is highly desirable that it should be performed quickly, safely, and with very little loss of blood; and that the wound should be so trivial that it should not add to the after dangers of the chief operation, and should not unduly protract convalescence.

Instruments.—After trying various instruments, I believe you will find these to be the best: a scalpel, a strong-pointed curved needle on a handle, a straight, sharp-pointed steel director, a blunt-pointed director, a tracheotomy dilator, the ends of which should be smooth and not bulbous. The laryngotomy tube should have a collar like the collar of an ordinary tracheotomy tube, and should be introduced on a solid guide, which projects well beyond the end of the tube and terminates almost, but not quite, in a point.

Operation.—A transverse incision about two thirds of an inch long is made over the crico-thyroid membrane through the skin and into, but not deeply into, the subcutaneous tissue. This just exposes some of the subcutaneous vessels, but does not wound them. The curved needle is thrust carefully through the membrane, as near the middle as possible, and between veins, if they are seen. On this the straight director is thrust through the membrane, as on a guide. The needle is withdrawn, and the blunt director is run through into the larynx in the groove of the sharp director. The two directors are forcibly separated, so as to allow of the introduction of the dilator when they are withdrawn. The opening is dilated, and the tube and guide are pushed through the opening, either before or after the dilator is withdrawn.

Laryngotomy, thus performed, rarely occupies more than a minute in the doing. There is usually no bleeding. The tube is withdrawn as soon as the chief operation is completed. The wound scarcely needs any dressing, is healed in the course of three to five days, and leaves scarcely a perceptible scar.

Accidents during Operation.—Two annoyances or accidents have occurred to me in the course of laryngotomy—hæmorrhage, and the breaking and thrusting back of a portion of the cricoid cartilage. Some of the first instruments which

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I employed were too sharp on their borders, and cut the thin-walled veins, or they were torn with the director. But, with care to keep close to the middle line, the danger of hæmorrhage is very small. If it occurs and is abundant, the wound must be opened a little wider, and the vessels exposed, and clamped, and tied; even this is trivial. On two occasions I thrust a strong, sharp-pointed director through what I believed to be the crico-thyroid membrane, for it passed deeply and smoothly, although it required some force to drive it in. On each occasion I was struck by the absence of exit of air through the director groove, and was very much puzzled to understand what had happened. In the hospital one afternoon this occurred, and I opened up the wound to discover the reason, for it seemed to me that it might lead to a serious complication. I found that the director had split off a piece of the cricoid cartilage, which was calcified, and had thrust it back, with the soft parts attached to it, into the larynx. From that time I began to use a needle on a handle as a guide to the sharp director, and I chose the curved needle, because it is difficult to employ it with sufficient force to break off a portion of the cricoid, and yet it can be used with very great precision.

The first tracheotomy dilators were bulbous; but the space for the introduction of the tube is so limited that there is only just room for the tube and very narrow ends of a dilator. The removal of the dilator after the introduction of the tube was attended with so much difficulty that the laryngotomy tube was several times badly bruised and dented; hence the change in its form.

It happened several times during the earlier operations that, after the sponge had been placed at the back of the throat, the patient's breathing became seriously embarrassed. Yet the tube was in place. This happened with the old laryngotomy tubes, which are not provided with a collar. It was soon found to be due to tilting up of the lower end of the tube, which threw it against the back wall of the larynx, by which it was quite blocked. The provision of a broad collar has almost entirely prevented this, and when it has occurred, a pair of clamp forceps fixed to the collar has sufficed to hold it in its place. The difficulty would also be obviated by the use of a longer laryngotomy tube.

Accidents after Operation.—After the operation, the only trouble I have seen has been subcutaneous emphysema, which happened in two or three very stout patients. The opening of the external wound lapped over the internal wound before the internal wound was closed, and air passing through the internal wound was driven into the subcutaneous tissue instead of escaping through the external wound. The introduction of a piece of gutta-percha tissue through into the larynx rapidly put an end to the annoyance.

If you perform operations on the tongue and jaws, on the palate and tonsil, or in the naso-pharynx, I cannot recommend this preliminary laryngotomy too strongly to you. It not only largely diminishes the danger of such operations, but it enables the operator to perform his operation deliberately, and with almost the same facility and comfort as if the disease were in an open part of the body. Think what this means when the disease is a malignant tumour, where it is of vital importance to be sure that the incision is carried into the healthy tissues far beyond it. Consider what a relief it is to both the surgeon and the anæsthetist to be able to work at the same time and at separate points, neither embarrassing or interfering with the other. When I began to perform this preliminary operation I am not sure that Mr. Gill, than whom there is no one in this town more skilled in administering the anæsthetic in a grave case of operation in the upper air-passages, did not look on the procedure with some contempt, for he knew how well he could keep the patient under an anæsthetic in very difficult cases. But both he and I are now quite in accord on the value of preliminary laryngotomy, and, if he were here, I am sure he would recommend it to you as strongly as I do now.

Of course, if you use it, put your best and most careful work into it, and use it with intelligence, otherwise it may play you false and will fall into disrepute. It scarcely seems reasonable to urge that the sponge which is placed at the back of the throat should be provided with a stout string or piece of tape, or to say that you must be sure that it is thrust well down to fill the lower pharynx and cover the larynx, or to warn you if it rises up into the mouth in the course of an operation, and is full of blood, that it must not be pressed back again, but must be replaced by another sponge, otherwise a quantity of blood will be pressed into the very channel from which you are so anxious to exclude it. I would not mention such errors had I not seen them quite or almost made.

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WITH SIR DYCE DUCKWORTH

AT

ST. BARTHOLOMEW'S HOSPITAL.

MAY 16th, 1902.

(*Concluded from p.* 100.)

THE next case I want to talk about is a boy who is too ill to bring here, who came in on the 9th April. He is a printer's boy fifteen years of age, and he is covered with a rash over most of his body, a rash which, after inspection, I am satisfied to call erythema multiforme. There are many varieties of erythema, diffuse in some cases, discrete in others, limited or very extensive and spread all over the body. He came into Matthew Ward with the skin eruption, and a temperature of 100·2°. He remained in that febrile condition for three or four days, and then the temperature began to rise considerably at night. After he had been in a week the temperature rose to 104·6°, and then was irregular, as you will see by looking at some of these charts. There are great flights of temperature, going up to 104° and 105°, and many of these high flights were accompanied by rigors. As I have said, our first impression was that he was suffering from erythema multiforme. You will remember I devoted one of my recent lectures to a consideration of erythema multiforme, and this patient appeared to present another example of this condition. But in course of time the boy seemed to get more and more ill, and the degree of pyrexia made one think the proper diagnosis had not been made. Erythema multiforme is a comprehensive term which is made to cover a great many conditions which have not yet been thoroughly investigated or brought into order; but probably all of them are due to some peccant or irritant matter in the system, a toxin introduced from some source. We were led by the character of the eruption to suspect that there was a rheumatic taint at the bottom of it, because erythema is the typical skin eruption of rheumatism. My first impression, then, was that it was rheumatism of the skin, manifested by this peculiar eruption. Therefore we listened carefully to the heart-sounds, and found a murmur which seemed to be confirmatory of the nature of the disorder. However, these flights of temperature and rigors did not seem to be compatible with the ordinary history of rheumatism. However severe rheumatism may be, it is not usual to meet with rigors. The heart changes did not make much progress, though the murmur remained. There were hardly any joint symptoms, though there were some; he had effusion into one or two joints, especially into the right knee; and again about the ankle there came a redness and puffiness. Those points seemed to indicate that the disease was of a rheumatic nature. Still, putting all things together—the high temperature, its sudden flights, the rigors, and the heart mischief—it seemed more likely that the patient was suffering from an infective endocarditis.

Owing to the doubtful nature of the case, I requested Dr. Gee to see him with me on one occasion, and while admitting the difficulty of the case, he thought the more recent diagnosis I had come to was the right one, or at all events was certainly justifiable. He agreed that it was very likely an endocarditis, with discharges into the general system of blood containing infective material, probably from some septic source, and that source most probably was the endocardium. Now we know that all forms of endocarditis are toxic, and that micro-organisms are to be found in all varieties of that disease. The simplest form—the rheumatic, for instance—is now believed to be due to a toxin, and a definite micro-organism has been sufficiently often recognised to make us feel confident that rheumatic fever is due to the *Diplococcus rheumaticus*. It seems to have very close relations indeed with the streptococci. So much is this the case that the bacteriologists are still in dispute as to the exact nature of it. That, however, is a dispute into which we cannot enter from the clinical point of view; it remains for laboratory research and for bacteriologists generally to settle that point. Meantime we are confident in believing that all forms of endocarditis are due to micro-organisms, however simple they may be. And it is a very interesting matter to know that definite micro-organisms at times vary in toxicity; that is to say, sometimes they are fairly benign, while at other times they may be very virulent and malignant, so much so as to destroy the patient. The discoverers in this country of the rheumatic microbe, Drs. Poynton and Paine, have recently published a communication which is partly clinical

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and partly bacteriological, which justifies them in believing that the ordinary rheumatic microbe is capable of this malignancy at times, and so, starting in its simplest form, may pass on to induce that variety of grave infective endocarditis which we so dread. The present case is interesting as having presented symptoms at first in the skin, then effusion into the joints, with those flights of pyrexia and rigors which are so grave and alarming. The patient remains exceedingly ill, and one feels very anxious about him, and as to what will be the result in the near future. Of infective endocarditis there are at least two varieties, one of which has been called the typhoid variety, which simulates very closely in its features enteric fever; the other is the intermittent and more chronic variety, which simulates more particularly septicæmia. Cases of this disease have often been overlooked. I do not suppose it is a new disease, but it has not been long recognised. In old days the cases were believed to be instances of continued fever,—that is to say, fever going on from week to week. The older physicians did not recognise a definite cause for the flights of temperature. The facts which we know were not known to them. In any case of the kind it is always well to have the blood tested for Widal's reaction. Grünbaum, of Liverpool, is believed to have discovered the Widal reaction before Widal, so I think we should call it the Grünbaum-Widal reaction. We tried for this more than once, but it did not appear. But it has been noticed in other cases of infective endocarditis. These cases, however, have been confounded in modern times with enteric fever. The fever is of the continued variety, going on from day to day, higher at night than in the morning, and accompanied by enlargement of the spleen. In many cases there is a condition which is common in typhoid fever, viz. diarrhœa, the stools looking very much like those of typhoid fever. Small wonder, then, that it has been difficult to tell what the disease is, especially when there has been an eruption on the skin. Again, it has been confounded with acute general tuberculosis, for this latter disease sometimes presents many of the signs of infective endocarditis. The enlargement of the spleen is mainly due to infarction, the emboli starting from diseased valves which have been damaged by the pre-existing rheumatic fever, such valves appearing to be a suitable nidus for the micro-organisms to grow upon. Healthy valves are less likely to be vulnerable in this way than damaged ones. Infective particles are shed from the endocardium sometimes into the brain, giving rise to cerebral symptoms, but more commonly into the spleen. At other times particles are shot off into the renal arteries, and give rise to infarction in the kidneys, which you recognise by the patient passing blood in his urine. In these cases there is pain in the loin associated with hæmaturia.

While still in doubt it is proper to ascertain whether any microbe can be detected in the blood. We requested Dr. Drysdale to examine this patient's blood on different occasions, but the result was negative. In previous cases of endocarditis I have had these examinations made. Dr. Kanthack, in one case, made an examination and discovered micro-organisms in the blood, and on that and other experiences was led to declare that any patient in whom infective organisms were found circulating in the blood was doomed to die. Streptococci are most commonly discovered. These examinations by Dr. Drysdale were both negative; nothing grew out of the blood. A swelling having occurred in this boy's knee the other day, we asked Dr. Drysdale to take some of the effusion from the left knee-joint, which he did. But here again the result of cultures from it was negative. Judging by Dr. Kanthack's *dictum* which I have mentioned, that fact seems so far to be of favourable augury in the case. Whatever vicious microbes there may be in this boy's system, then, they cannot be found by the ordinary methods of detection. He has had no enlargement of the spleen, his skin affection is much less than it was, and his temperature in the last few days has rather declined. These are rather in favour of a hopeful view.

The treatment of these cases is generally to sustain the patient's strength by good food, by quinine and iron. But in all these cases, as a rule, drugs have been ineffectual. We have tried in this case what benefit we can give by administering quantities of yeast, which is believed to have an effect in destroying these germs in the blood. No very decided effects have so far been observed from this treatment. The most modern treatment is by antistreptococcic serum, and that is now generally

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practised in these cases. We have used it on several occasions here. The results were not remarkable in any way. One is told that it is necessary to inject every twenty-four hours five to twenty cubic centimetres. We have watched the effects of this serum given antiseptically and carefully. The serum for the purpose is to be obtained now from various sources, and we know that it is prepared from a mixed series of cultures. Some physicians have used this method very frequently, but have not often had definitely good results from it. I do not believe that it has ever done any harm, but it does not appear to be very efficacious. But the principle is in accordance with the modern idea that you may find an antitoxin to neutralise everything of a specific nature circulating in the blood. Perhaps we ought to have used this serum more assiduously than we have done. Meantime this boy is still in a precarious state, and the prognosis must remain doubtful. It is perhaps too much to hope that he will get well. The majority of these cases die, but not all. You will see from time to time in the journals cases reported in which the diagnosis may be regarded as correct, having been made by careful men, and in which the patients have recovered. The disease may be very acute and carry off the patient in a few days, or it may go on, as in one case I had, for twelve months. That was an intermittent case, the subject being a woman in John Ward. She ultimately died. Meantime our great object is to maintain the patient's strength and try to neutralise the effect of the poison. The heart symptoms and its murmurs alter; one day you will notice a different note from that on another, showing some alteration in the mechanism, probably due to fragments having been shot off. This disease is more frequently found on the left side of the heart than on the right side, and in the latter case you are very apt to have septic plugs projected into the lungs, and you will recognise that fact by the patient spitting up blood. Localised gangrene may be subsequently produced at the spot.

(May 29th.—Since this lecture was delivered the patient has continued in much the same condition. Daily exacerbations of fever occur towards evening, but the rigors seldom arise. There are no signs of embolism, and the joints remain free from pain and effusion. The appetite remains good. There have been some fresh erythematous patches over the trunk and limbs. He is now taking twenty minims of oil of gaultheria thrice a day, and continues the yeast.)

CERTAIN USES OF ELECTRICITY IN DISEASES OF THE NERVOUS SYSTEM.

By LEONARD S. DUDGEON, M.R.C.P.Lond.,

Assistant in the Electrical Department, St. Thomas's Hospital; Pathologist and Registrar to the East London Hospital for Children.

(*Concluded from p.* 112.)

General Treatment.

Electricity does not hold a very high place in therapeutics for the treatment of diseases of the nervous system. There are some medical men, however, who profess to have seen beneficial results from electrical treatment in nearly every disease known to clinicians. We have only to examine certain text-books which deal with electro-therapeutics to prove this statement. One of the greatest advantages of this method of treatment is due to the considerable psychological effect which it produces, not only with patients suffering from functional disease, but in nearly every known disorder. There are but few diseases in which it has not been tried, and generally stated with beneficial results. Most patients are greatly impressed with electrical treatment, even if it is doing more harm than good; they usually only see the bright side of it. Those disorders in which it offers any curative treatment are extremely few, but there are many diseases of the nervous system which do receive temporary benefit. The method of treatment in each case will be described as fully as space will permit.

The varieties of electro-therapeutics are as follows:—(1) Galvanism; (2) faradism; (3) static or franklinic electricity; (4) the sinusoidal current.

Galvanism may be used either (*a*) locally or (*b*) generally.

There are two methods of local galvanisation;* (*a*) unipolar, (*b*) bipolar.

(*a*) Unipolar. The active electrode is placed over the affected area, the inactive elsewhere.

(*b*) Bipolar. Both electrodes are placed over the affected area.

* Monell, 'The Treatment of Disease by Electric Currents;' quoted by J. H. Bryant, 'Text-book of Pharmacology and Therapeutics,' edited by W. Hale White, 1902, p. 988.

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Stabile and labile galvanism may also be employed; the latter is the more common. At St. Thomas's Hospital we generally use labile galvanism, and the kathode is the active electrode.

The strength of the current should vary, for adults from 5 milliampères to commence with up to 15 and 20, for children from 3 milliampères with gradual increase in strength. It is frequently recommended that a current of so many cells should be used for electrical treatment; this statement is absolutely valueless, no certain calculation could be made from it. All current strength should be recorded in milliampères. It is generally sufficient for the patient to receive ten to fifteen minutes' treatment, in some cases three times weekly, but generally every day if possible.

Faradism is used in the same way as galvanism.

Electrodes.—There are numerous electrodes for therapeutic purposes in every-day use, whilst enormous numbers on the market are seldom seen. Besides the testing electrodes which for therapeutic purposes are sometimes useful, we have larger but similar kinds to these, also wire-brush electrodes of various shapes, so useful in functional disease, sponge electrodes, etc. The diameter of these should vary from one to three inches; usually a two-inch is most satisfactory.

The Electric Bath.—At St. Thomas's Hospital we treat most of our patients by means of the electric baths. The common varieties of these baths are—(1) the arm bath; (2) the hip bath; (3) the full-length bath.

The arm bath is generally used for lead palsies, peripheral neuritis due to various causes,—in fact, every lesion of the nerves or muscles of the upper extremities; also for such diseases as acroparæsthesia, Raynaud's, and erythromelalgia, etc.

The hip bath is used for similar palsies of the lower extremities to those described above, and for sciatica.

The full-length bath is used for neurasthenia, sciatica, etc. These baths are usually constructed of porcelain. Two copper plates are placed in the bath, at the foot and head; the current therefore can only enter the body through the water. The current may be the constant or the induced, and may be derived from a galvanic battery, an induction coil, or from alternating electric-lighting circuit (Hedley).

In the monopolar bath, one electrode is placed in the bath of water, the other is applied to some part of the patient's body without contact with the water; this pole may be either anode or kathode.

The full-length galvanic bath should be commenced at about 50 milliampères, and the strength of the current increased as desired. The faradic bath is generally supplied with the current from the main, which is regulated to the requirements for medical purposes by passing through a transformer. About fifteen minutes to half an hour's immersion is usually quite sufficient. The temperature of the baths should be about 95° F. These general baths undoubtedly greatly improve the patient's general condition.

Local electric baths are similar to the full-length bath already described. For most cases fifteen to twenty minutes' immersion is usually sufficient, and the baths should be given daily, but not less than three times weekly.

The sinusoidal current is recommended by Bordier* and others in place of the ordinary faradic and galvanic baths in cases of lead palsy, etc. Lewis Jones claims that the advantages of the sinusoidal currents are mainly—(1) the great smoothness with which the current varies; (2) the great magnitude of the currents which can be borne.

General faradisation and galvanisation are recommended by Beard and Rockwell, but faradisation is much pleasanter and much more commonly used, as it is said to give much better results.

Static Electricity.—This form of electricity, which was so commonly used in the past, has now come into general use, and it is most highly recommended by Monell† for the treatment of numerous diseases. The electricity yielded by static machines is of very high electro-motive force. The number of volts can be estimated by the length of the sparks, frequently amounting to 100,000 and more. The strength of the current is, however, extremely small. For general or local franklinisation the patient is placed in an insulating chair. For general effects one pole of the static machine is connected with the chair from ten to thirty minutes; for local use, an electrode is connected with the other pole, and this is brought near the

* 'Professor Bordier, 'Précis d'Electrothérapie,' 1897, pp. 43 and 168.

† Monell, 'The Treatment of Diseases by Electric Currents.'

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region which is to be treated. Two forms of electrodes are usually used, one is fitted with a point, the other with a ball; in the latter case sparks are able to be produced. Generally it is stated that the negative pole has a depressing influence on most people, while the positive pole has the opposite effect. The positive pole can be recognised from the negative when a static machine is in action by means of the flame of a lighted candle; this is directed towards the positive pole.

Treatment by static electricity produces important results. Bryant* says it causes increase of body-weight, increase of oxidation, an increase of the amount of urine, increases appetite, increases the excretion of urea, lowers blood-pressure, promotes gentle perspiration, diminishes the excretion of uric acid, and stimulates the function of nerves and muscles. Sedative effects are best produced by the positive breeze, lasting from five to twenty minutes. Counter-irritant effects can be produced by spark applications.

ELECTRICAL TREATMENT OF SPECIAL DISEASES.

Infantile Paralysis.—If the muscles respond to the faradic current it can be used, but the galvanic current is certainly better, and some state that faradism is harmful and increases the tendency to atrophy.

This treatment should be commenced as soon as the acute stage is over, generally in about three or four weeks from the onset of the paralysis. If the galvanic current is used, two flat electrodes should be attached to the wires of the battery, the negative and active electrode being placed on the affected limb. The current should be only of sufficient strength for the patient to withstand, and sufficient to cause muscular contraction. With children it is usually better to commence without any current passing through the electrodes, as they become more accustomed to the condition of affairs as time advances, and electrical treatment becomes much easier as soon as they understand what really is taking place.

At first galvanism should be used for about ten minutes three times a week; later it should be used every day if possible. Some electricians recommend placing a flat electrode to the cervical portion of the spinal column, *e. g.* in paralysis of the arm. This idea arose from the belief that the current passed to the spinal centre.

If both legs are completely or only partially paralysed, it is a good plan to place them in a bath in which the strength of the faradic or galvanic current can be regulated, as already explained. Electrical treatment should be continued for at least one year. The friends of the child can always use the hand battery, and there is seldom any fear of too strong a current being applied.

What is the value of this treatment in these cases? This disease is accompanied by nerve-tissue degeneration, and we know that muscles whose nerve-fibres degenerate undergo degenerative changes and finally perish if there is no nerve regeneration. It is obvious that muscles in such cases cannot receive any stimulus by their nerve-fibres, but if we supply a stimulus by means of electricity, it will be found that the sensitiveness of the paralysed muscles is increased both to voluntary and electrical stimulation. This is sometimes found to be the case, as muscles which do not respond to galvanism at the first or second attempt may react at the end of a week, and a certain amount of movement may also take place. The value of electricity is found to be chiefly for those cases where there has been only damage to the nerve-cells and fibres. Its sole use is to prevent muscles from undergoing degeneration, so that if the nerve-cells and fibres regain their power to transmit stimuli, the muscles which will receive these impulses may be as near normal as possible. In those cases where there is absolute destruction of the nerve-cells, etc., electrical treatment is useless. It is important to remember that in this disease more than in any other, the value of electrical treatment has been greatly over-estimated; but, on the other hand, many children with infantile paralysis might receive considerable benefit from massage and electricity if this treatment were only commenced as early as possible, before the muscles of the affected limb have completely degenerated, which is so often allowed to take place. Lastly, we have here no better illustration of what has been already stated, that because certain muscles in recent cases fail to respond to either faradism or galvanism, we should not allow them to absolutely degenerate, but we ought to treat these muscles just as carefully as those which respond to the electric current. This does not seem to be fully understood; many people

* J. H. Bryant, loc. cit., p. 996.

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think if the reaction of degeneration is obtained, or no response at all, that therefore a certain muscle or group of muscles must perish; this is quite incorrect. It may safely be said that improvement may take place up to a period of two years.

Hemiplegia.—Electrical treatment cannot produce a cure, but it is said to be of great value during the period of recovery, or in the post-hemiplegic state. Dr. Lewis Jones * remarks, "The electrical treatment favours the return of functional activity in those parts which have been disused or impaired, though not destroyed by the cerebral lesion." Electrical treatment should not be commenced till about four weeks from the onset of the attack. Those people to whom this treatment is recommended should have the galvanic or faradic current so as to produce distinct contraction of the muscles three times weekly for about fifteen minutes. This treatment should be continued for about six weeks. It is always well to remember that any very startling results must never be expected; the uses of electricity are always most limited in diseases of the upper motor segment.

Infantile Hemiplegia.—The same remark applies here to electrical treatment as in adult hemiplegia. I have not seen any advantage from this method of treatment, except in one case. A child æt. 2 attended the electrical department, under Dr. Turney, and received faradism daily to the affected limb; the final result was excellent. Of course, one cannot be certain that electrical treatment produced this effect, as massage was also given twice daily. For the rigidity which follows a hemiplegia, electricity is sometimes of service to relieve this complication. Either faradism or galvanism may be used.

Facial Palsy.—A weak galvanic or faradic current may be recommended for the treatment of the paralysed muscles due to peripheral lesion. We never use electricity for this deformity at St. Thomas's Hospital, as massage to the face has produced such excellent results in some of our cases, while in others there has been no local application used, with quite as good a result. Theoretically, if electricity is useful for one peripheral lesion, it should be for all, and, any way, it should keep the muscles stimulated, if it does nothing else. A weak galvanic current should be used daily, only just sufficient to cause muscular contraction. On no account should faradism be used if there is any evidence of contraction. Very often, even with a weak current, there is considerable pain when the electrode is applied to any motor point of the nerve.

Lead Palsy.—This is one of the most typical examples of a nerve lesion improving under the influence of electricity. Most peripheral nerve lesions do well when treatment is commenced as soon as possible, but cases of lead palsy more especially. Possibly this may be due to the fact that these patients do not generally have degeneration of all their tissues, which is so common with patients suffering from alcoholic neuritis; also those cases of lead palsy which improve are generally less severe than in other examples of peripheral neuritis. Of course, in the advanced cases electrical treatment often has little effect.

In the electrical department of St. Thomas's Hospital all cases of lead palsy which has involved the arms are treated with the faradic bath for about ten minutes, at first three times weekly, and after a short period for fifteen minutes daily, gradually increasing the strength of the current. In those cases in which the legs or body generally are involved, a full-length bath is advisable. Dr. Lewis Jones * recommends the sinusoidal current and the arm bath for most of his cases of wrist-drop. The results in early cases, as already stated, are excellent. Even in long-standing cases with marked atrophic change in the muscles, etc., there is often some improvement, but not to any considerable extent.

For those unable to have an electric bath, the ordinary faradic or galvanic battery does fairly well, but the bath treatment should always be used if possible. Many cases completely recover, but I am sorry to say as soon as they are well, or even improved, they resume former work, with the natural result that relapses are common.

Peripheral Neuritis.—For these cases it is generally better to use galvanism. The strength of the current should vary from 5 to 20 milliampères, according to the condition of the patient, and the active and negative electrode should be applied to the affected muscles in the form of labile galvanism.

* Loc. cit., H. Lewis Jones.

* H. Lewis Jones, 'Dangerous Trades,' 1902, p. 375.

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This treatment must not be commenced till the acute stage is over. At first it should be used for fifteen minutes three times weekly; afterwards daily for the same time or a little longer. Other people recommend the sparks of a static machine for chronic cases. Sinusoidal or galvanic baths are also of much value.

Progressive Muscular Atrophy.—Electrical treatment is recommended by some physicians. Patients for whom we have used this method of treatment have claimed that they feel much better; but, especially when we consider the pathology of this disease, we must suspect that the improvement is purely subjective. If electricity is used, only a weak galvanic or faradic current should be applied to the muscles, so as not to produce fatigue; or, still better, the electric baths for fifteen minutes three times weekly.

Sciatica.—No electrical treatment should be used during the acute stage of sciatica, otherwise severe pain may be produced. In the later stages patients should take hip-baths daily for ten or fifteen minutes, or the faradic current may be used along the course of the nerve. If galvanism is used, the positive electrode is placed over the sciatic notch, the negative and active electrode is traced up and down the leg in the direction already mentioned; but to relieve pain the kathode is used over the sacrum and the anode is applied to the tender spots, using a current varying from 5 to 10 milliampères. In some cases electrical treatment is of very considerable value, while in others this treatment is a dead failure. Monell* strongly recommends static electricity for these cases. The patient being seated on the insulated chair, connect the shepherd's crook† with the negative pole, and instruct the patient to hold it with both hands; ground the positive pole and the brass point electrode, then move the electrode over the painful area, holding the point two or three inches from the part. At the end of the sitting hold it nearer, so as to obtain a few sparks. At first the treatment should be used daily, later on every other day.

Localised Palsy.—In paralysis of the ulnar, median, or musculo-spiral nerves, electrical treatment is of very great value, either faradism or galvanism. In cases of long standing there is generally some improvement, even if it is only of slight amount.

A boy under the care of Mr. Clutton suffering from external popliteal paralysis improved completely under the influence of faradism and massage daily. It is important to remember what has been previously stated, that the electrical reactions may show partial or complete R.D., but the power of movement may have returned. This particular case of popliteal palsy was an excellent example of such a condition.

Myelitis.—Where atrophy is present, galvanism or faradism should be used so as to keep the muscles in tone. No such treatment, of course, should be used during the acute stage.

Functional Disease.—Electricity is one of the most important additions we have in clinical medicine to establish a diagnosis in certain cases, and in some of these cases a little stronger current will complete the diagnosis and the treatment at one and the same time. No better example can be shown than the case of a man who came to the out-patient department of St. Thomas's Hospital with complete loss of power in the left arm. There was wrist-drop, and also some weakness of the ulnar muscles. This man used a crutch on the left side, owing to the loss of his left leg. I diagnosed this as a case of crutch palsy. Three days afterwards he was seen by Dr. Turney. There was now well-marked spasm in the fingers, quite a new feature, and on careful consideration of the case Dr. Turney diagnosed functional palsy. The electrical reactions were taken, and were found to be normal. A wire brush electrode was afterwards used in place of the testing electrode, with a very strong faradic current. Such an excellent result was obtained, that in about five minutes diagnosis and treatment were complete. This case shows most clearly how a mistaken diagnosis may be corrected by taking the electrical reactions, and how treatment may be combined at the same time. While, on the one hand, physicians in charge of electrical departments at general hospitals, etc., frequently see patients who are benefited very rapidly by this method of treatment; yet, on the other hand, many advanced cases are absolutely unaffected for some time, although they have the battery daily for a considerable period. For example, a German tailor came

* Monell, 'The Treatment of Diseases by Electric Currents;' quoted by J. M. Bryant, loc. cit.

† This is a rod for connecting either pole of the machine with the platform on which the chair rests.

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to the electrical department of St. Thomas's Hospital with great regularity for five weeks, always with the same statement, "I have paralysis of my arms and legs." He improved on each occasion on the application of a very strong faradic current, but it was only after the fifth sitting that this result was permanent. This is very frequently the case, and patients suffering from paralyses may do well at the time, or for a short time afterwards, but the result may be only temporary, relapses frequently occurring. A point of great importance is always to remember that before violent electrical treatment of any kind is adopted, we must be certain that the case is of functional origin; if otherwise, much damage may be done. There is no doubt also that electricity produces a considerable amount of mental impression. This can be illustrated occasionally by increasing the vibrating power of Wagner's hammer, which is used with the magnet to produce the interruptions of the faradic current; the louder the noise produced by the hammer, the more effect it has. Static electricity, especially if large and numerous sparks are produced, generally gives excellent results, or, according to competent observers, still better results may be obtained by using "the rain of sparks."

Functional Aphonia.—Elaborate intra-laryngeal electrodes have been invented for the treatment of this disease, but they sometimes produce more harm than good. Excellent results can be obtained by placing the broad, flat electrode to the back, and stroking the wire brush connected with the faradic current up and down the skin over the larynx. A gradually increasing strength of the current may be employed; but others recommend that we should commence with a very strong faradic current, with the hope that the patient will shout "Oh!" These cases, although they frequently relapse, are cured for the time being, frequently after the first sitting, but sometimes a considerable period may elapse before a complete cure is effected.

Gastric Neuroses.—A special electrode has been devised for introduction into the stomach in intractable cases of this disease, but there can be very few physicians who could possibly give much credit for the results obtained. The only case in which it has been used at St. Thomas's Hospital within the last year it was found to be absolutely useless.

Neurasthenia.—Professor Strümpell* summarises the electrical treatment in this disease as follows:—"It is hard to say whether all these methods of electrical treatment have any specific action. The greatest part of the benefit obtained is undoubtedly of a suggestive nature; but there are few other methods by which such a suggestive effect can be so easily produced as by electricity." General faradism can be employed. For this treatment the patient is almost entirely stripped, a large flat electrode is placed in connection with the feet, and a wire-brush electrode is used to sweep all over the body. (For full details see "General Treatment.") Many cases do well by simply using the induced current from a small battery.

Raynaud's Disease.—The affected limb should be placed in a bath of warm water, more especially an electric arm bath, so that the patient will receive benefit from the diffusion of the current in the water, especially in the region of the copper plates. If an electric arm bath cannot be procured, a basin of water and a constant battery will do almost as well, the negative and active electrode is placed in the bath, the positive and inactive on the upper part of the arm. The affected limb should be kept in the bath from fifteen to twenty minutes, if possible, during an acute attack. Those patients who have come to St. Thomas's Hospital have generally been greatly benefited by this method of treatment. It should be used whenever there is an acute paroxysm, and after each paroxysm, daily for twenty minutes, for some few days. Of course in any case in which there are fissures, chilblains, or trophic lesions, etc., on the fingers, bath treatment is generally contra-indicated. Sir Thomas Barlow† strongly recommends this method of treatment in acute cases. In chronic cases he advises galvanism, shampooing, and Swedish movements daily.

Erythromelalgia.—The same treatment may be applied as in Raynaud's disease.

Tubes Dorsalis.—Electricity is of little value in the treatment of this disease. It is, however, recommended by certain eminent authorities, either in the hope of some temporary improvement, or to check, if possible, the progress which this disease makes.

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* Professor Strümpell, loc. cit., p. 1199.

† Sir Thomas Barlow, "Raynaud's Disease," Allbutt's 'System of Medicine,' vol. vi, p. 605.

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Galvanism is recommended by Erb,* Oppenheim,† and others. We place a large, flat, and inactive electrode to the patient's neck, and the other to the lumbar region. The strength of the current should vary from 5 to 10 milliampères. It should be used daily, if possible, for about five minutes, and must be continued for some months. As I have previously stated, it is doubtful whether this treatment is of much value. For the severe pain, such a common symptom in tabes, Monell‡ prefers static electricity to all other forms. He uses the positive spray with mild sparks, and the brass-pointed electrode. For the headache, the positive breeze; for numbness and anæsthesia, sparks. Incontinence of urine in tabes is treated by internal faradisation of the sphincter vesicæ with good results by Professor Oppenheim.§ Other authorities recommend galvanism to the perineum or over the symphysis, with a current strength varying from 10 to 20 milliampères.

Neuralgia.—Local treatment in those cases which are suited for electricity often produces good results. Faradism should be applied to the affected nerve or nerves in gradually increasing strength, using a flat electrode, or by the application of the wire brush to the skin over the affected area. This latter method may cause much pain before any improvement occurs. Stabile galvanism is also used, the inactive electrode or anode is applied to the painful spots or to the affected nerve-trunk, often with good results. We must, however, regulate the strength of the current carefully. Faradic or galvanic baths are equally useful in suitable cases. Static electricity may be equally beneficial, and should be used in the manner described under trigeminal neuralgia. Lastly, I know of a case of severe neuralgia in a lady which ceased temporarily either by a full-length sinusoidal bath, or the electric-light bath. We may justly say that electricity often gives marked temporary benefit in neuralgia, but very rarely produces complete cure.

Trigeminal Neuralgia.—Galvanism is usually recommended in those cases in which electrical treatment is advisable. The anode should be applied to the painful points of exit of the affected nerve, and to other places where pain is felt; the kathode is placed at the back of the neck. It is better to use only weak currents and to gradually increase the strength. Those cases which respond to this method of treatment will generally improve more rapidly if it is combined with facial massage. Others use cataphoresis. Monell, as usual advises static electricity. The positive breeze, which acts as a local sedative, should be kept in motion over the affected area. Pain is often said to disappear in a few minutes, but the treatment must be continued for a little longer. This should be combined with general electrical treatment, and both should continue for about ten days to a fortnight. In counter-irritation a spark should be used.

Professional Neuroses.—Electricity is quite useless in these cases unless the patient will take absolute rest from work. Galvanism is generally most suitable, either galvanic baths daily for about ten to fifteen minutes until there is complete recovery, or a local application with a curren varying from 5 to 15 milliampères. Strong currents, however, should be avoided. Faradism is sometimes recommended in very mild cases without much spasm; static electricity is also useful. Personally, the cases which I have seen at St. Thomas's Hospital have benefited mostly from the galvanic bath, combined, of course, with massage, which is possibly the more useful of the two methods of treatment.

Cataphoresis, or Electric Osmosis.—If the platinum terminals connected with a galvanic battery are placed in a bath with a porous partition, so that water fills one side and saline solution the other, rapid osmosis will take place if the anode is placed on the side in which there is plain water, and the kathode on the side of the salt solution; but if we reverse the poles, the osmosis will be much slower and in the opposite direction. This electric phenomenon has been made use of in medicine, especially in France and America. Various drugs in solution (iodide of potassium, cocaine, strychnine, etc.) may be introduced into the patient's body through the unbroken skin. The skin acts as a medium between its own body fluids and the drug in question. Strychnine poisoning has been produced in this way; also when potassium iodide has been similarly administered, it has been found in the saliva.

Lastly, I must thank Dr. Turney for permission to mention any facts relating to the patients attending the electrical department of St. Thomas's Hospital.

* Professor Erb, loc. cit.

† Professor Oppenheim, loc. cit.

‡ Monell, quoted by J. H. Bryant, loc. cit., p. 1003.

§ Professor Oppenheim, loc. cit.

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THE CLINICAL JOURNAL,

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* *Specially reported for The Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE
ON
SOME CASES ILLUSTRATING THE DIAGNOSIS OF TYPHOID FEVER.

Delivered at Guy's Hospital, October 4th, 1902.

By W. HALE WHITE, M.D., F.R.C.P.,

Physician to the Hospital.

GENTLEMEN,—About a month ago there were admitted under me almost simultaneously three cases thought to be typhoid fever, and, as in each there was considerable difficulty about the diagnosis, I thought we should be profitably occupied to-day if we considered the points about them. Most of you have seen all three of the patients. In the first that we will take, the difficulty lay between a diagnosis of typhoid fever and appendicitis. The patient was named Mary W—, and she was æt. 14, and she came on September 3rd on account of pain in the abdomen and headache. She told us she was very liable to headaches. On August 28th she had to come home from school because she did not feel well, and after she got home she was sick and complained of pain in the abdomen. The sickness soon stopped, but as she remained ill she was brought to the hospital. On admission her temperature was 101·2°, her pulse was 120, her abdomen was somewhat distended, and her tongue was rather suggestive of typhoid fever. On September 1st, that is to say the next day after admission, she was much the same. The temperature all day remained at about 102°, and she still complained of pain across the abdomen, but it varied very much in position. I saw her on the afternoon of the next day, September 2nd, and I was told that some thought she was suffering from typhoid fever, while a small minority considered the case to be one of appendicitis. Her temperature had remained up since admission, her tongue

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had the same appearance as it had when she came in, the abdomen was slightly but uniformly distended, and she still complained of some pain across it; but the position of the pain was very variable, and the pain itself was not at all severe. She had not been sick since admission; she had a quiet pulse, and her headache was better. No abnormal dulness or a sense of rigidity or tumour could be made out on palpating the abdomen, and although it was generally slightly tender the tenderness was not in one part more than in another. A rectal examination revealed nothing abnormal. I expressed the opinion that the patient was suffering from appendicitis, for a careful examination of the rest of the body revealed no cause outside the abdomen for her pyrexia, and it seemed to me that the sudden onset, the pain, and the general slight abdominal tenderness, the sickness and the constipation were all in favour of appendicitis. The patient appeared comfortable, with a good pulse and moist tongue, and so it was decided to wait for the examination of the blood. The next day on coming to the hospital Mr. Greenfield, my house physician, told me he had seen the patient at noon and she was then very comfortable, with a good pulse, and her condition was quite unchanged from what I had seen on the afternoon of the day before. At 2 p.m. I went to the ward—that is to say only two hours after Mr. Greenfield had seen her—and found her with a small pulse, feeble and rapid, a rigid abdomen, an anxious drawn look about the face, and her temperature since Mr. Greenfield had last seen her had fallen 3 degrees, that is to say, 3 degrees in two hours. It was clear that during the last two hours she had developed acute general peritonitis. I then heard that two blood-counts had been made since the afternoon of the day before. The one of the evening of the day before showed 40,000 leucocytes to the cubic millimetre, and the one on the morning of the day we were then in showed 21,000 to the cubic millimetre. It was clear, therefore, as the leucocytosis was observed before the general peritonitis came on, that it could not be attributed to that; and, further, such excessive leucocytosis showed clearly, as again it had been observed before the general peritonitis came on, that the case was not one of typhoid fever, for you know that if there is any alteration, as there usually is, in the number of white corpuscles in a case of typhoid fever, it is always in the direction of a diminution from the normal; a count of about 5000 is a common count to get in cases of typhoid fever. Therefore the diagnosis on the previous day of appendicitis was confirmed and extended, for inasmuch as very marked leucocytosis had been observed before the general peritonitis came on, it was clear that the appendicitis was associated with an abscess, and also we were able to interpret what had happened, for the diagnosis now clearly was that we had a patient suffering from appendicitis with an abscess (a high leucocytosis count showing an abscess), and, further, we learnt that within the last two hours this had led to acute general peritonitis. Mr. Fagge saw her at once, and, agreeing with the diagnosis, operated the same afternoon, and found the diagnosis was quite correct, for he found the patient had very early acute general peritonitis, as early as it is ever seen at an operation. And further, he found a large abscess behind the cæcum in connection with the appendix. So you see here we had a case which was thought by the majority of those who watched her during the first few days of her stay in the hospital to be a case of typhoid fever, but which turned out to be a case of appendicular abscess; and inasmuch as this is the second time this year that I have known the difficulty between the two to be great and to be raised, I thought we might take that for the first case, and go over with you how to tell a case of appendicitis from one of typhoid fever.

Now let us go over the points that we had in connection with this case. The first thing we had was undoubtedly a sudden onset. You will say that is very rare in typhoid fever. I agree with you; it is very rare. When it does occur it is usually associated with a chill. Two years ago I remember a lecture which Dr. Taylor gave you on chills in typhoid fever. Our patient had no chill. The usual onset in a case of typhoid fever you know is for the patient to gradually become ill. For the first day he thinks he is a little out of sorts; the next day he thinks he is a little more out of sorts, as the text-books tell you, the feeling of general malaise increases during the first week, at the end of which he usually consults a doctor. Our case was therefore atypical in the mode of onset; it began in a way that typhoid fever very

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rarely begins; on the other hand, it began in just the way that you would expect a case of appendicitis to begin, for, as a rule, in acute appendicitis the onset is sudden.

The next point to which I would direct your attention, as helping to make a diagnosis, is the pain. Now there is no doubt whatever that the majority of patients with typhoid fever do not complain of any abdominal pain. You must remember that whether or not you get a history of pain depends very largely upon the care with which you cross-examine the patient. I think if you were to say to me, "Can you be sure, quite sure, that you have had no abdominal pain during the last few days?" I should find it difficult to reply in the negative. So you must take a distinct and undoubted history of pain as being of varying value to you; and a distinct and undoubted voluntary complaint of pain on the part of the patient is the exception in typhoid fever. Nevertheless it may, in a few cases, occur. So in this particular patient the fact that she voluntarily complained of pain—you will remember the pain was so bad that she came home from school because of it, so it was of considerable intensity—would make you suspect that the case was not one of typhoid fever. Another thing is this: even if the patient with typhoid fever does complain of pain it is not usually such a severe and continual pain as our patient suffered from. What I think misled those of you who thought she was a case of typhoid fever was that the pain was not localised to any particular spot. That I quite grant you was misleading. There is no doubt that in this girl the pain was sometimes in one part of the abdomen and sometimes in another. Nevertheless severe abdominal pain should put you on your guard against diagnosing typhoid fever, for in typhoid fever it rarely occurs. Then you will remember that this girl was tender in the abdomen. Now there is no doubt that tenderness in the abdomen may occur in the course of typhoid fever, but it is exceptional. Considerable tenderness, as this girl had—or perhaps we might be more accurate and say "some tenderness"—should make you suspect that your diagnosis of typhoid fever is incorrect. On the other hand, tenderness is a common symptom of appendicitis. But what put the men who thought the case was typhoid fever off the right scent was that the tenderness was not, in this particular patient, limited to any particular spot.

The next point you will remember with regard to her was that when she came home from school she was sick. You will ask, can typhoid fever ever be ushered in by sickness? Undoubtedly it can; and what made it especially difficult to interpret correctly in this case is, that in children it is particularly ushered in by sickness. Children vomit very easily. Almost any specific fever may, in a child, be ushered in by vomiting. Still, a distinct history of vomiting, such as this, was undoubtedly in favour of appendicitis as against typhoid fever. It is uncommon in typhoid. The tongue, as was pointed out to me, did look like that of typhoid fever. You all know the typical tongue, which is clean on the tip and edges and furred on the dorsum, and perhaps a little clean in the middle line. But I think that many exaggerate the value of the tongue as a diagnostic sign of typhoid fever. I quite grant you that this is the common condition of tongue. Sometimes, however, in typhoid fever, the tongue is uniformly furred, and here we had a case in which the patient undoubtedly did not have typhoid fever, and yet the tongue was suggestive of that disease.

The last thing which this patient complained of was headache. I am a great believer in the importance of headache as a help to the diagnosis of typhoid fever; but in this girl you will notice how quickly the headache went. Headache in typhoid fever is usually a dull, long-continued, weary, wearing headache; it is often mostly over the front of the head, and you know it will continue most commonly for some days, often until the tenth or the fifteenth. The headache is so severe that frequently the patient complains of nothing but the headache; and when it is so severe as that I do not know anything which will relieve it better than potassium bromide. But you will remember that in the history of this girl the headache very soon passed away, and this feature of it was, I think, evidence that it ought not to be regarded as a headache due to typhoid fever, but merely as a febrile headache, as the subsequent history of the case showed. So the reasons which led me to side with the minority who thought it was a case of appendicitis were, that there was a sudden onset, that there was considerable abdominal pain, considerable abdominal

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tenderness, that there was some vomiting. I discarded the condition of the tongue and the headache, because the latter soon passed away, and consequently we came to the conclusion, and wrote that conclusion up over the bed, that the case was one of appendicitis. On the first afternoon that she was seen many of you expressed surprise that there should be difficulty in the diagnosis between typhoid and appendicitis, and I quite admit that many books do not mention it. For instance, since this case was in I naturally referred to Nothnagel's 'Encyclopædia,' which occupies very many volumes. There is an article on typhoid fever occupying nearly an entire volume, and the difficulty of diagnosis between typhoid fever and appendicitis is not insisted upon, and, indeed, it is hardly mentioned. But if you look at that wonderful repository of clinical observation, Fagge and Pye-Smith's 'Medicine,' you will find the difficulty is referred to; and the difficulty which is there mentioned is also alluded to in Osler's 'Medicine.' So I think you can fairly bear the difficulty in mind. But I observe that most authors put the difficulty rather the other way round. We had a case which the majority thought was typhoid fever, but which turned out to be appendicitis; but I notice that the prevailing difficulty is that the case is one of typhoid fever and is thought to be appendicitis. Ours was not quite the common difficulty. I need hardly mention, before we leave this subject, that of course books mention that you may have great difficulty, when perforation has occurred, in telling whether perforative peritonitis is due to typhoid or to appendicitis, but that did not enter here. There was no question of perforation having occurred when we first saw the girl, and I doubt very much whether it matters when you diagnose when perforation has occurred whether the perforation is following upon appendicitis or typhoid, for in either case of course the only thing is immediately to do a laparotomy.

Now you will say that as our patient had got acute general peritonitis too early in the disease for perforation in typhoid fever the blood-count would have led to a correct diagnosis. And, indeed, if it had chanced that a visit was paid to her by all of us in the morning of the second day I saw her we should have had nothing but the blood-count to go by, and that would have been conclusive. So it behoves us to look at the difference between the blood-count in appendicitis and the blood-count in a patient with typhoid fever. In typhoid as regards the red blood-corpuscles there is, as you would expect in long-continued severe fever, a slight diminution, both in the number of reds and in the amount of hæmoglobin. From day to day the diminution is so slight as to be hardly noticeable, but at the end of a long illness like typhoid it is marked. But the striking thing, as our other cases during the autumn have shown—and the men who have been round with me have seen it demonstrated in the wards—about the blood-count in typhoid, is that it is one of the few specific fevers in which there is leukopenia; that is to say, the number of white cells gradually diminishes as the illness goes on, and as few as 1000 per c.mm. have been recorded. When you remember that the normal number of white cells per cubic millimetre is somewhere about 10,000 you will see what a falling off there is in white cells where you get a count of only 1000. And if in a case of typhoid fever you have, instead of this diminution of whites, an excess, you may be certain that you have to deal with a complication. And the commonest complication causing leucocytosis which you have to deal with is phlebitis. So if the white cells have been low and they suddenly mount again, there is likely to be phlebitis associated with the typhoid fever, or you may have perforation. Contrast all this with appendicitis. In appendicitis there is no time for the fall in the number of red corpuscles and the slight fall in the quantity of hæmoglobin to attain any striking proportions. But, on the other hand, unless the case is very slight—when it will not be confounded with typhoid fever—or unless it is like some of these fulminating cases, so severe, and kills so quickly that there is no time for the reaction to occur, that is to say unless the case is excessively severe or trivially slight, there is always an increase in the white cells in appendicitis. And when you get up to numbers as high as we did in this case, 21,000 and 40,000 per cubic millimetre, you may be almost certain that your appendicitis has become complicated by the presence of pus, and therefore it is of the very greatest importance in determining about operation.

The second case which afforded some difficulty in diagnosis was a boy, who was admitted about the same time. George J—, æt. 8, was admitted

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on August 31st into Philip Ward, bed 16. On Thursday, August 28th, he first complained of being ill, with severe headache. On August 30th he was much worse, being delirious. On the 31st he was still worse and was admitted into the hospital, supposed at that time to be suffering from meningitis. September 1st was a Sunday, and I did not see him, but I am told by Mr. Greenfield that he still resembled a case of meningitis. I saw him on Monday, September 2nd, and then all of you that were round with me noticed that he lay with his knees drawn up; he was curled up in bed, his head was markedly retracted, he appeared to be suffering much from headache, his abdomen was not particularly distended; he much resented interference, so much so that I remember he made us smile by the instantaneous way in which, when we attempted to examine him, he curled himself up still tighter and dragged the bedclothes over him when we took them down. His temperature and pulse were both raised, there was a doubtful history of vomiting, and at that stage it was an open question what was the matter with the patient, whether it was typhoid or whether it was meningitis. In fact, the evidence up to that point was considerably in favour of meningitis. But we continued our examination of him and found one single spot, which was a fairly typical typhoid spot. We went over him still further and noticed that we could feel the spleen easily, which indicated that it was enlarged. Later on the Widal reaction was obtained with his blood, and the next day or so a blood-count was made, and his white cells were found to be down to 4500, that is to say about half the normal. At the same time when we were going round we looked at his optic discs, but what we learned from them was rather unsatisfactory, because they were just faintly blurred at the edges. Here, therefore, is a capital case for us to discuss, for it was one which was regarded as meningitis, but it turned out to be one of typhoid fever.

You will notice that what incidentally settled the diagnosis in that case was the presence of a single spot, and I think you will agree with me that I am right when I point out to you so constantly at the bedside that one of the most dramatic things in medicine almost, is that from the presence of a little spot, often not much bigger than the head of a large pin, we are able to prophesy that the body of that patient is teeming with typhoid bacilli, and if at that moment we were able to make a post-mortem we should find that Peyer's patches were enlarged. And if ever you find people inclined to jeer at you, and to scoff at the ability of doctors, tell them that fact, and I think they will at once admit that there is something in diagnosis after all. And it was that one spot which enabled us to tell precisely what was the matter with the patient. So I think it is clearly of great importance that we should for a moment consider exactly what a typhoid spot is. You all know that these spots are called rose spots, a term which is so ingrained that it passes; but I object to it because roses vary in colour, and it is rare to see a rose exactly of the colour of a typhoid spot. But you know the pale red tint of the typhoid spot. They are small, they are more or less round, they are circumscribed, they hardly ever run together, they vary in size from that of a pin-head to that of a good big pea; and you may see a man in Philip Ward now with very large typhoid spots. It should be pointed out that they are slightly elevated, and, what is very important indeed, the colour disappears on pressure. A hæmorrhagic typhoid spot is one of the rarest things in medicine; most of us die without ever having seen one. So if your spot is hæmorrhagic it is almost certainly not typhoid but typhus which you are dealing with. They usually appear somewhere in the second week, from the seventh to the tenth day, and I was pointing out to you in the ward that they begin mostly on the abdomen and trunk, and if they are on the extremities they are less marked than they are on the trunk. So if you showed me a case which you thought to be typhoid, and the spots were more on the extremities than on the trunk, I think it would be a good reason for doubting the correctness of your diagnosis. You know that the average life of a spot is about three or four days, and you know that typhoid bacilli can be cultivated from these spots. Usually there are somewhere between five and thirty in the same patient at the same time, and over 80 per cent. of cases of typhoid have these spots.

I have left to the last on purpose what is the most important feature of typhoid spots. There is no doubt, I think, that the most characteristic feature of typhoid spots is that they come and go in crops. Over and over again you will

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turn down the bedclothes and see some spots which you find it difficult to be sure whether they are typhoid spots or not. Put a ring round them, so that you may know where to find them, and come again in two days' time; look at the abdomen. If those which you have put a ring round are gone, and others have come up, it is almost certain you are dealing with a case of typhoid fever. It is the coming and going in successive crops which is unlike most other exanthemas; for instance, the scarlet fever rash is all out and done with it; measles is the same; but the typhoid fever spots come in successive crops, and that is the great point to remember about them. As a rule, the rash lasts about fourteen days, but it may, as I have pointed out, in some cases come on long after the patient is convalescent, and we have had such cases here.

Although of late years many other points have come to help us to diagnose typhoid fever—for instance, the diminution in the number of white cells, the clumping of typhoid bacilli that we commonly call Widal's reaction, the diazo reaction, and the fact that occasionally a person with typhoid fever will show typhoid bacilli in his urine,—yet every one of these is of small importance compared with the spots. No one of these points which I have mentioned to you is more valuable than the spots, and spots are, for all practical purposes, perfectly infallible. Ever since I have been at Guy's I have only heard of two cases in which spots which reputable observers mistook for typhoid spots were found in association with other diseases, and neither of those cases has occurred under my own care. So that after some years in a hospital where typhoid fever is a very common disease, one has not seen a single disease in which spots resembling those of typhoid fever have occurred when the case was not one of typhoid. In spite of the many new modes and great helps in the diagnosis which have come up of late years, the spots remain by far the most important sign, and their presence saved us in this particular case. We did not have to wait for the Widal reaction test, or for the blood-count; from that one spot we were able to say that the case was one of typhoid fever.

With regard to the other things which help us, the Widal reaction often helps us very much. It is just as well that I should take the opportunity of telling you how we stand with regard to that. The weak part about it is that while a considerable number of cases of typhoid fever, over 90 per cent., show the Widal reaction, its advent may be delayed. It is rarely present before the seventh or tenth day, and its advent may be delayed after that. So if you have to deal with a sign the advent of which may be delayed till after the second week, a negative examination is not of the great help which you might hope it would be. And then in many parts of the world there would be practical difficulty in applying the test. You will not be perhaps near a cultivation of typhoid bacilli to see whether the blood from your patient clumps them or not. You will say "What about the other way round? You said the Widal reaction is nearly but not quite always present, that is to say it is present in over 90 per cent. You have pointed out that the weak part of it is that its advent may be considerably delayed; but does it invariably follow that if clumping occurs the patient has got typhoid fever?" We had in Guy's not long ago a case bearing on that admirably. A woman came to Miriam Ward, and the diagnosis lay between malignant endocarditis and typhoid fever occurring in a patient who had a murmur. That is a common difficulty which we will not consider to-day. When she came in her blood was tested for the Widal reaction, and the result was negative. There was no clumping to be obtained with the proper dilution in that case, either with 5 per cent. or 0·5 per cent. dilution of the serum. That is very important in connection with what I am going to tell you, because many people's blood-serum clumps because they have had typhoid fever years before. The previous time when I had taken the Clinical Ward, we had a woman in the ward whose serum gave Widal's reaction, but she had not got typhoid fever. Then on cross-questioning her we found she had typhoid fever twenty-three years before. The longest period during which the reaction persisted which I have known was that of a patient whose serum continued to clump typhoid bacilli after twenty-six years. So that nearly every case that is published of some other disease than typhoid agglutinating the typhoid bacilli, does not withstand the criticism that the patient may have had typhoid fever before. If you record a case of a man with pneumonia whose blood clumped typhoid bacilli, it is worth nothing unless you can show that he had not had typhoid fever for many years,

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which is often difficult. But our patient's serum did not clump when she came in, and gradually the diagnosis became undoubtedly one of malignant endocarditis. Widal's reaction was again tried after she had been in some time, and then her serum gave it. On one occasion her serum clumped with four out of five strains, some of them with 0·5 per cent. dilution, so it was, as regards Widal's reaction, as typical a case of typhoid as you could want. That aroused our interest very much, because we had come to the conclusion then that it was not typhoid fever but malignant endocarditis. And it could not have been that she had had typhoid fever previously, because she did not clump when she came in. We tried to get typhoid bacilli from the urine, but none could be found in it. When she died from malignant endocarditis, and cultivations were taken from the spleen, there were no typhoid bacilli in it, and she had neither during life nor after death anything but Widal's reaction to suggest typhoid. So here was the case of a patient whose clumping could not be due to the fact that she had typhoid fever, a case of clumping due to a different disease, namely, malignant endocarditis. But such cases are excessively rare. Nearly always if there is clumping it means the case is one of typhoid fever. That, I think, is the position of our knowledge with regard to the Widal reaction.

The blood-count and the spots I have already mentioned to you.

Now you will say, "Really, is this difficulty between meningitis and typhoid of much importance?" It has not turned up lately, I think, but it is quite important. To show you that it is important, a question on it was set at the M.B. examination in 1879. The question was, "How would you tell a case of meningitis from one of typhoid fever?" I thought I would bring you some evidence that the matter is of importance. Sir Samuel Wilks says, discussing tubercular meningitis, "In the first place the child may be fretful, listless, have stomach disturbance, and other troubles which bear no especial characters; afterwards feverish or pyretic symptoms, marked by hot skin, quick pulse, furred tongue, and headache. At this period the case is generally, I might say always, styled one of gastric (*i. e.* typhoid) fever. It is not the exception, but the rule, to make a mistake; the stomach disturbances are so common, and these cerebral affections commence so insidiously, that the severe nature of the case is not suspected. I believe, in the majority of cases to which I am called, the case is regarded as one of fever in which cerebral symptoms have become developed; and I think, with the utmost acumen which you can use, you cannot positively determine the disease at this early period. Some time ago I was asked by a more than ordinarily acute practitioner to see a young man who, having been ailing a few days, was causing some anxiety to his friends. He had febrile symptoms, and the question, as in all such cases, was whether these arose from a local inflammation or not. We could discover no cause, and were therefore obliged to style the case one of fever. We were both alive, however, to the possibility of tubercle formation, and of early meningitis, but failed to discover any proof of either. On the next day there was some suspicion of head affection, and on the following day there could be no doubt of its existence. You will see that the diagnosis of continued fever is as often made for negative reasons as for positive ones. A person is in a highly febrile condition, and if a characteristic rash is at the same time present, the nature of the case is apparent; if there is no rash we look for some local inflammation to account for the symptoms; if none be discovered, we have again recourse to the term fever.

"Since, then, at the commencement of tubercular meningitis the head symptoms are not more marked than in ordinary fever, a positive diagnosis cannot be made. I am justified in this opinion by having to-day looked over my note-book, used when I was a student, and reading the particulars of the case of a child who was in the hospital under Dr. Addison, to which are appended some notes of his clinical lecture. He alluded to the difficulties attendant on the diagnosis of tubercular meningitis and gastric fever, and after commenting on the case, stated his belief that the symptoms present denoted a cerebral affection which would be necessarily fatal. It so happened that on the very next day these head symptoms passed off, and the child got rapidly well." So I think we may fairly say that if Addison in his day gave a clinical lecture pointing out the difficulty, and Wilks also in his, it is a genuine difficulty that we have to face.

The other points besides those mentioned a few

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minutes ago which might help to a right conclusion are these:—Headache is more severe in meningitis. If it is so severe that the patient is holding his head, as children do, and the headache is evidently almost more than the child can possibly bear, then the case is probably one of meningitis, and not typhoid. On the other hand, the typical headache of typhoid fever is a dull headache, an aching pain which disappears after some days, not later than the fifteenth.

Next as to delirium. Our child upset the commonly received notions about the delirium. But, as a rule, the typhoid patient is not delirious nearly so early as the meningitis patient. You know Jenner's point, which was a good clinical point, that in typhoid fever when headache disappears delirium begins, but in meningitis delirium and headache are often co-existent. Then again, delirium in typhoid fever is not, at any rate early in the case, nearly so severe. It is true that cases of typhoid fever are recorded in which a patient has jumped out of bed and run about the ward quite delirious, but they are the exception. Very severe delirium early in the case is an evidence of meningitis. Patients in both the diseases may become comatose, but in the case of typhoid fever only comatose towards the end of the disease, when the patient is sinking into the typhoid state, and then the diagnosis has usually already been made. Other things being equal, coma comes on earlier in meningitis, and is, generally speaking, much more profound in meningitis. Both patients may have their mental faculties dull, both may be dull and apathetic, but in a general way the typhoid patient is not so dull and stupid as the meningitic patient. Cases of meningitis also typically resent all interference. It is true that in this and many other points our typhoid patient resembled meningitis, but this only shows how great the difficulty may sometimes be. But, as a rule, the typhoid patient does not markedly resent this interference, and in many cases he seems rather proud of being able to show his spots. The meningitis case will drag the bedclothes up over him as fast as you take them down.

Then retraction of the head is a great help, that is to say in meningitis, as opposed to typhoid. Children have a habit, which is from the point of view of diagnosis very provoking, of getting retraction of the head in all sorts of conditions. Remember that, as in our case, there may be severe retraction of the head, and yet the case may be one of typhoid fever; nevertheless retraction of the head is a point in favour of meningitis as against typhoid fever.

With regard to the pupils. The condition of them may be a great help. I do not think the pupils are ever unequal in typhoid; and if anything they are dilated. Unequal pupils would make me almost certain that the case was one of meningitis; and so, I might say, would a minutely contracted pupil. Again, the optic discs are of great importance. Definite optic neuritis is conclusive, I think, in favour of meningitis. Some years ago I was particularly interested in the point, and for a long while my house physician and I looked systematically at all the discs in typhoid fever. In any considerable fever you may get a little extra redness of the disc, but in typhoid fever it never goes on to striking blurring of the margin and actual swelling. Definite optic neuritis would be in favour of meningitis. But you will notice that the earliest stage of optic neuritis is a little redness, so as in our case when all that strikes you about the disc is that there is a little extra redness, you cannot say for certain whether you have before you a very early optic neuritis; in such a case an unequal redness of the discs is a great help.

With regard to irregularity of respiration and pulse, either or both are much in favour of meningitis as opposed to typhoid fever. The reason is the same as that for the irregularity of pupils; in both cases it is probably due to the exudation at the base of the brain unequally affecting the nerves on the two sides. A slow pulse is in favour of meningitis. Vomiting may occur in both diseases, but it is much more frequent in meningitis than it is in typhoid fever.

Passing down the body to the abdomen we come to retraction of the abdomen. Retraction of the abdomen is a symptom the importance of which for the diagnosis of meningitis you cannot over-estimate. Three years ago a man lay in No. 2 bed in Philip Ward, whose condition was diagnosed by Dr. Shaw as meningitis solely on account of retraction of the abdomen, and the diagnosis turned out to be correct. On the other hand, the abdomen in typhoid fever is usually distended. But in this child it was neither the one nor the other. The

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spleen is very rarely enlarged in meningitis, but an enlarged spleen is considerable evidence in favour of typhoid fever.

Convulsions I ought to have mentioned under the head of the nervous system. These may occur in children with typhoid fever; but persistent and repeated convulsions, when you are in doubt which the case is, should make you tend rather towards meningitis than towards typhoid.

The tongue we have already discussed, and the stools are not of much importance. As a rule, the patients with meningitis are constipated, and so are many patients with typhoid fever. But sometimes you get the so-called pea-soup stool of typhoid fever, but its importance is enormously exaggerated. You say, "Can I find the bacilli?" A few years ago we thought that typhoid bacilli could be easily recovered from the urine; but that, probably, is an error. They do occur in the urine, and it was held that if they could be cultivated from it, it would be proof that the patient was suffering from the disease. But, as Denyer showed in his thesis published in the Guy's Reports, it is very difficult to find typhoid bacilli in the urine of undoubted cases of typhoid fever.

So much for the diagnosis between the two. I have not alluded to the difficulty of diagnosis that may occur towards the end, namely, that a person with typhoid fever often, towards the end of the disease, sinks into a low muttering delirium. There is much subsultus, and he is comatose. He is also obviously enfeebled in his mind, and grinds his teeth and has delusions. All that occurring towards the end of the case is on a different footing, and I only mention it to warn you that meningitis must not therefore be assumed to have complicated the typhoid fever. It is virtually an unknown complication; these symptoms merely mean that the patient is desperately ill from his typhoid.

The last case I wish to bring before you to-day I can only mention very briefly. The patient was admitted almost on the same day as the other two. His name was Thomas C—, æt. 15, who came into Philip Ward on August 30th. While walking out four days before, he felt sick and giddy, and had diarrhœa. The next day he had abdominal pain, and as this continued he came to the hospital. For the last two days he had been constipated at times. His temperature was 99° and the pulse 74, and so practically he was healthy with regard to the temperature and pulse. He looked fairly well, and was regarded by those who admitted him as a case of gastro-enteritis, and he was given five grains of calomel. On the 31st his temperature was 102°, and the bowels were open. On September 1st the temperature was 100°, and as he was sweating profusely he was regarded as suffering from rheumatic fever, and accordingly was given salicylates. On September 2nd the spleen was palpable, but there were no spots. On September 4th I saw him, and we turned him over and looked at his back, and again a spot saved us from error. There was one spot over his right scapula. Later fresh spots developed, and later still he showed the Widal reaction, and we found that the white cells were 7200 per cubic millimetre. So you see this was such a slight case that if it had not been for the single spot the diagnosis would have been overlooked, because it was the single spot which instigated us to try the Widal reaction and to do the blood-count. That patient illustrates how difficult may be the diagnosis when the case is very slight. Not long before that I had seen with Dr. Landon a case of persistent headache in a woman who was otherwise so well that she had been bicycling twenty miles a day, and had regarded herself as almost well, and had stopped bicycling only because of the headache. The temperature was normal when I took it myself, but bearing in mind the fact which we have dwelt upon about headache, we turned her over and found again, oddly enough on the back, a single spot. It was during the few hot days we have had this summer, and as there was only this single spot it was very difficult to be sure that the spot was not due to the excessive perspiration. So we left the diagnosis of typhoid fever till another spot came. It did come, and so the case was proved to be typhoid fever. Here, therefore, are two cases showing that the person may have a normal temperature, and yet the presence of a spot may make the diagnosis of typhoid fever certain.

WE have received from Messrs. J. and A. Churchill a copy of 'Cancer and other Tumours of the Stomach,' by Dr. Samuel Fenwick and Dr. W. Soltau Fenwick. The volume presents many points of interest, and will be duly noticed in this Journal.

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WITH DR. E. W. AINLEY WALKER AT GUY'S HOSPITAL.

General Pathology of Infection.—I.

By the term *infection* we mean the entrance into the tissues of living micro-organisms. If those micro-organisms are capable of living and multiplying in the invaded tissues they produce disease. Such disease is called *infective* disease; and the micro-organisms which give rise to it are said to be *pathogenetic* micro-organisms.

If the infective agent is either continuously, or from time to time, or again at certain definite periods discharged from the body of the infected subject—in the secreta or excreta, by desquamation from the surface or otherwise—the disease will be *infectious* as well as infective; that is to say, it will be transferable from the infected to the healthy subject,—as, for example, in diphtheria. The organism may then be spread about—disseminated—by air, by water, or by food, and cast-off clothing and the like, if it be capable of a continued existence outside the living body under ordinary conditions. Or, if it be incapable of growth and life except within the tissues or secretions of the body the disease, of which it is the causal agent, will be *contagious* only. That is to say, it will be only capable of spread by contact transference from the diseased to unaffected individuals,—as in the case, for example, of gonorrhœa.

Contagious diseases, then, can only be acquired by contact with the affected subject, or with quite fresh infective material from his body; while in the other infectious diseases the contagium vivum or virus may reach the unaffected individual through the agency of air or water, or some other means of spreading.

The distinction which I drew just now between the terms infective and infectious must be clearly borne in mind from the outset in the discussion of infection. The former term implies that *micro-organisms* are the *causal* agents in the disease in question; the latter simply that the disease is *transferable*, and therefore tends to appear in epidemics. Some infectious diseases have been proved to be infective; in a number of others there is considerable evidence of infective origin, though exact proof is lacking; while in the rest the direct evidence is at present altogether wanting, though from analogy they are assumed to be infective also. And this assumption forms a working hypothesis, which we shall find convenient for our purpose here.

That certain diseases are infectious had been known from very early times, but the cause of their infectiousness remained entirely undetermined. An infectious disease was seen to be endemic in one region, in another only epidemic; to spread from the endemic centres along lines of travel, becoming epidemic in its course, now in one place, now in another; to pass with almost lightning rapidity from individual to individual, and from town to town; to die away again, only to be followed after months or years by a new outbreak, and again another. It was seen to favour centres of insanitation and of over-population; to vary in severity in successive epidemics and without apparent cause; to undergo seasonal variations where endemic; to pass by touch, by the breath, by food, by objects handled by the sick, it might be only months or years before, and even to follow the exhumation of bodies of those long dead of the disease. But the cause of its infectiousness remained unknown.

All the evidence, however, already pointed in the direction of a particulate infective agent; and, indeed, of one able to multiply, and that with great rapidity and almost without limit,—in fact, of a contagium vivum. This was the view expressed in 1671 by Kircher, who employed the phrase contagium animatum. It had long been foreshadowed in the opinion of the old Roman writer Varro, who attributed marsh fevers to the bites of small invisible flies. And you will remember the still more ancient theory that a particular evil power, by name Beelzebub, was specially solicitous for harmful flies, and was their god.

Paracelsus also came quite near the truth in his conception of what he called the "seeds" of a disease, an expression which still clings in popular language. But it remained for Leeuwenhoek (1675 on) to lay the first foundations of microbiology by his discoveries in the microscopic world. Other investigations followed, and new facts were ascertained, and gradually a natural history of the unicellular plants developed; but it was long before their causal agency in the decompositions of organic substances was determined. Conclusive proof of this was first supplied by Pasteur in his work on

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the phenomena of fermentation and of putrefaction, published in 1857.

It was Davaine who was the first to clearly recognise the *rôle* of micro-organisms in disease. He proved the fact for anthrax. Then Villemin, in 1865, following the earlier experiments of Klencke (1843), found that he could produce tuberculosis in animals by the inoculation of tuberculous material; and came to the conclusion that the virus of this disease was comparable in its mode of action with those of other infective diseases. This conclusion was strenuously opposed from many sides, but was confirmed by Armanni in 1873, and by Cohnheim and Salomonsen. The nature of the tuberculous virus itself, however, remained unknown.

In 1876 it was shown by Burdon Sanderson that in erysipelas the fluid taken from the spreading edge always contains large numbers of micrococci arranged in chains; and Ogston found, in 1881, the constant presence in acute abscesses of what he termed the cluster coccus; thus demonstrating the definite association of certain kinds of micro-organisms with particular diseases. In 1882 the discovery of the tubercle bacillus was announced by Koch, and shortly afterwards he published his extended series of masterly researches on tuberculosis, and made known his culture methods, which at once elevated the study of bacteria from the position of a natural history to that of natural science.

By the application of these methods to the postulates laid down by Koch, our knowledge of bacteria and of their relation to infective processes has rapidly increased, and a new science of Bacteriology has been developed. In its relation to bacteria in general, their forms and characters and general biology, the science of Bacteriology is a branch of Botany, and of no greater interest in medicine than any other of the natural sciences; but as concerns the relation of bacteria to disease, the study of the action of micro-organisms in the living body, and the reaction to their growth and metabolic products, forms an important section of the science of Pathology, and is of constantly increasing value.

Of the micro-organisms at present known which produce disease in animals, only a very few belong to the animal kingdom, notably the parasites of malarial fevers; the vast majority are vegetable micro-organisms, and are usually, though somewhat inaccurately, spoken of as bacteria. They belong to the Mycetes.

The Mycetes are subdivided into the following groups:

1. Hyphomycetes, or Moulds, which are composed of a mycelium bearing hyphæ, by means of which the reproductive processes are carried on.

2. Blastomycetes, or budding fungi, in which the new members are budded off from the parent cells.

3. Schizomycetes, or fission fungi, which multiply by fission.

To the first class belong the actinomyces, or ray-fungus, and the moulds associated with certain skin diseases, such as favus and ringworm. *The second class* possesses few if any pathogenetic species, though certain pathogenetic torulæ have been described. While the *third class* comprises the great bulk of the micro-organisms of disease. For the special description, general biology, and distinctive characters of these bacteria I must refer you to your lectures in bacteriology, or to Muir and Ritchie's text-book; they do not especially concern us here.

In their relation to the tissues of the living body, the micro-organisms may be divided into the parasitic and the saprophytic groups; parasitic being such as can live and develop in the living tissues, while the saprophytic can live outside the living animal in dead animal tissue, or on vegetable substances, or even on inorganic matter. Each of these groups may again be subdivided into two, giving the four following classes:

Parasitic forms.—1. Obligatory parasites, which can *only* live in living tissue. 2. Facultative saprophytes, which *can* continue to live outside the living body.

Saprophytic forms. — 3. Facultative parasites; which, while normally saprophytes, *can* exist in living tissue. 4. Obligatory saprophytes; which *cannot* exist within the living animal.

The chief importance of this method of classification lies in its bearing on the question of infection, and on the distinction between such diseases as are *infectious* in the general sense, and such as are *contagious* only in the narrower meaning. The micro-organisms concerned as causal agents in the latter or exclusively contagious infections belong entirely to the first class of the obligatory parasites, which can *only* live under the conditions found in

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living tissue. This contact infection was spoken of by the late Professor Kanthack as "direct contagion," to distinguish it from what he denominated "indirect contagion." Diseases which can be "indirectly" contagious in Professor Kanthack's sense as well as "directly" contagious I have included under the general term *infections*, confining the term contagion to its literal sense of contact.

Those diseases in which the infective agent leaves the diseased subject in a condition in which it can *immediately* infect a healthy person I shall call directly infectious and directly contagious diseases respectively; reserving the terms indirectly infectious and indirectly contagious for those diseases in which the agent of infection is not directly transferable nor capable of directly reproducing the disease, but only mediately transferable through the body of an intermediate host.

This is merely a matter of terms, but it seems preferable to use the word contact in relation to the *subject* of disease, whether man or animal, rather than to the *causal agent* of the disease in question. Used in the latter sense the term directly contagious would have to be applied to all infections, since acquirement of the disease implies a direct contact with the infective agent and its entrance into the living tissues of the body.

It must not be supposed from what I have said of the obligatory parasites that their faculty of living within the living tissues, and only there, is in any way dependent on the fact that those tissues are alive, except in so far as this implies merely the presence of physical and chemical conditions not otherwise obtainable in nature, and which are necessary to their existence.

For, as we shall see later, there are continually in action in living tissue certain processes which are definitely and often markedly inimical to bacterial life. All that is meant is that it is only in living tissue that the bacteria in question can obtain those conditions of temperature, moisture, and nutrition in general which are essential to their life and growth.

In a number of cases we are already able artificially to provide conditions sufficiently similar, though still enormously removed from those which actually exist within the body, to enable us to cultivate bacteria which are or have been looked on as obligatory parasites, so that what is in nature an obligatory parasite can be grown in the laboratory on artificial media; while in other cases gradually increasing knowledge and skill have shown that organisms once quite impossible of cultivation or only grown with difficulty after prolonged and careful trial, and hence at first regarded as obligatory parasites, can actually exist and multiply outside the body under quite ordinary conditions. Thus the tubercle bacillus, which Koch only eventually succeeded in growing on inspissated blood-serum, and which took years to force to grow on agar, has actually been shown by Sander to be capable of growth on baked bread or boiled macaroni, in potato juice or ordinary potatoes, and even tap water under normal conditions; while certain of its allies have their normal habitat upon a common grass, the so-called quaking-grass.

It follows, therefore, that as our knowledge advances the class of the obligatory parasites is being continually reduced in number by the relegation of its members one by one to the group of facultative saprophytes; while, on the other hand, those which remain admitted as obligatory parasites in nature are proving more and more amenable to improved artificial culture methods under such approximation to their normal habitat as can be made in the laboratory.

The facts which I have here referred to briefly bear closely on the question of the spread of the infective maladies, and the means by which that spread may be prevented or at least checked. Regarded from this aspect these diseases may be divided into groups and classes more or less definitely separable from each other, according to the nature and life-history of the infective agent.

For the following division I am indebted partly to the late Professor Kanthack's article in Clifford Allbutt's 'System of Medicine.'

Group I, in which the infective agent never leaves the body of the infected animal, or does so only in a condition in which it is incapable of immediately infecting another animal of the same species. Examples of this are to be seen in the spirillum of relapsing fever and in the malarial parasites. Such diseases are only indirectly infectious after the passage of the infective agent through the body of an intermediate host. They are to be met either by removal from the region in which they are endemic, or by hygienic means directed to the destruction of the intermediate

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host. Isolation of the sick will not affect the spread of the disease.

Group II, in which the infective agent leaves the body of the infected animal in a condition in which it *is* capable of immediately infecting a healthy individual. Such diseases are directly infectious. They may be treated in the following classes:

Class 1.—Diseases due to obligatory parasites. In these the infective agent cannot continue to exist outside the animal body; they are accordingly purely contagious. Absolute isolation of infected subjects would eventually result in the total disappearance of the diseases in question.

Class 2.—Diseases due to facultative saprophytes. In these the infective agent can exist outside the living body, though it does not usually do so. Here again isolation will be of advantage; but the main object must be disinfection of the secreta and excreta of the infected subjects, since these contain infective agents capable of life and growth outside the living animal.

Class 3.—Diseases due to facultative parasites. In them the organism is a natural saprophyte. Prevention of disease can, therefore, only be attained by rendering its natural habitat no longer suitable. Isolation is of little preventive value in regions where the infective agent is disseminated as a saprophyte.

Those micro-organisms which are capable of invading the tissues of the body and there producing morbid processes are spoken of, as I said at the commencement, as *pathogenetic* forms. But besides these, the body surfaces, internal and external, are *normally* inhabited by a great variety of bacteria; some of these are capable under suitable conditions of taking on pathogenetic action,—as, for instance, the ordinary colon bacillus; others are invariably harmless under normal circumstances, and are pure saprophytes. Others again are possibly not merely harmless, but actually useful to the organism, especially such as have their normal habitat on the digestive surfaces. For it has been shown that plants, at any rate, do not thrive when grown on absolutely sterile nutritive material, and probably the same will be found true for animals. Such different forms of life as live together for their mutual advantage are spoken of as *symbiontic* organisms.

Pathogenetic bacteria are not sharply marked off from harmless forms by any well-defined division. This could not be expected; for the power of invading living tissue is a factor of two variables, and therefore variable itself. These factors are the activity of virulence of the bacterium in question, and the resistance of the organism concerned. Thus it happens that one and the same bacterium may be pathogenetic for one kind of animal but not for another of a higher resistance, as in the case of frogs, which are resistent against anthrax, and fowls to tetanus infection. Or the bacterium may be pathogenetic for a given animal at one time, but not at another, as the resistance of the animal varies; this is the case with tuberculous infections, or with infection by the *Bacillus pyocyaneus*. Again, the activity of the bacterium itself may vary, so that it is at one time pathogenetic, at another not so. Or it may be when taken by itself entirely harmless, but become capable of harmful action when associated with another, possibly equally non-pathogenetic organism. In the same way the action of a bacterium which is usually pathogenetic may be prevented or counteracted by its association with another organism. The meaning of many of these facts will become clearer when we consider the nature of the action of bacteria, and the reaction to them which occurs within the living body.

I have stated that all bacteria which are capable of continued life and growth in the living tissues are pathogenetic to the animal concerned, and by their presence and their products will inevitably cause disease. Certain remarkable observations have, however, been recorded recently which, if confirmed, go far to controvert this view. For though all previous observers who have worked on normal organs have declared the tissues of healthy animals to be invariably sterile, Ford, working under the direction of Professor Adami, claims to have found in rabbits, guinea-pigs, cats, and dogs, that not less than 70 per cent. of the organs of animals killed during normal health show the presence of living bacteria in their substance when examined in the usual way with all precautions. Those experiments, if confirmed, will very considerably modify our view of the extent to which symbiosis plays a part in normal animal life.

However this may be, the fact remains that the effect of the growth of *abnormal* organisms in the body tissues is to produce disease. If such disease

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presents a character and course such as distinguish it clearly from all other known diseases, and is invariably associated with the presence of a particular micro-organism, and no other, then the disease is spoken of as *specific*, and the infective agent which produces it is called the specific organism of that disease.

Professor Koch laid down the conditions of specificity for a bacterium in the form of his three famous postulates:

1. That the bacterium in question, and no other, is *invariably* present in the typical lesions of the disease concerned, and of *no other* disease.

2. That it can be *cultivated* from the diseased animal on artificial media through several generations. And—

3. That when suitably injected into healthy animals it gives rise to the *reproduction of the characteristic picture* of the disease, and can be *again recovered* from the lesions of the infected animal.

If these postulates are fulfilled the bacterial specificity of the disease is proved. It has been so proved for anthrax, diphtheria, tetanus, gonorrhœa, malignant œdema, plague, tuberculosis, glanders, and actinomycosis. Of the other infectious diseases, in some the proof is altogether wanting, while in the rest it is still incomplete. Thus in typhoid fever, influenza, and mycetoma (Madura disease) the successful animal experiment has not been hitherto established; though, as regards typhoid fever, both Remlinger and Chantmesse have produced a disease with intestinal symptoms and marked infiltration of the Peyer's patches in animals by means of the *Bacillus typhosus;* and I have myself, both in the rabbit and the guinea-pig, obtained by injections of the same bacillus a continued fever of some weeks' duration associated with variable diarrhœa, a loss of appetite, lassitude, and great wasting; and showing post mortem an ulcerated condition of the small intestine and cæcum indistinguishable from the typhoid ulceration seen in man.

In relapsing fever and in leprosy only the constant presence of a particular organism has been established, all attempts at cultivation having failed; though in relapsing fever it has been shown that the blood of diseased subjects is capable of reproducing the disease in monkeys when injected. In the case of leprosy experimental inoculation has entirely failed to reproduce disease in man wherever the subject was above suspicion of already harbouring the disease in latent form.

In a number of other diseases of infective origin which present clinically a definite identity no *one* bacterium can be said to be the causal agent, since a variety of different micro-organisms can be found in different cases, and sometimes more than one in the same case. Such are the pneumonias, cholera, all suppurative inflammations, endocarditis, meningitis, and the like.

Finally, there remain a number of infectious diseases which are as yet only assumed, not proved to be infective, such as rabies, syphilis, yaws, yellow fever, mumps, whooping-cough, and all the exanthemata.

This is the position of our present knowledge with regard to the *bacterial specificity* of the infective diseases. But we can go a step farther than this in the establishment of specificity. A pathogenetic micro-organism which gains entrance to the living tissues can only act upon them in one of the two following ways, namely, either by chemical or by mechanical interference with their functions. The second of these means is, in the vast majority of cases, of very slight importance, if of any. In very few conditions is the multiplication of bacteria so enormous as to produce a mechanical effect,—such, for example, as a bacterial embolism within the living animal; nor is the distribution of secondary foci of infection usually such as could be produced by a mechanical action of the micro-organisms. Even where the multiplication of bacteria is great enough to cause mechanical effects, these are in general overshadowed by the more important chemical action of the bacterial products. Such mechanical effects as do occur are usually secondary to this action. Thus emboli, which are only rarely, and then probably only in capillaries, formed entirely of masses of bacteria, are very frequently produced by fragments of thrombi formed under the chemical action of bacterial products on the blood and endothelium of vessels, and gradually softened and dissociated by the action of the micro-organisms which grow and multiply in them.

The pathogenetic action of bacteria accordingly depends almost entirely on their power of forming harmful substances, to which the symptoms and phenomena of disease are due. Hence we require, for complete demonstration of the specificity of any

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given disease, to prove not only its bacterial specificity, but also that of the bacterial products. This proof can only be obtained by separating the bacterial products and producing the disease concerned by their inoculation in the absence of the micro-organisms themselves.

Unfortunately this presents exceptional difficulties. The conditions under which the toxic substances are formed within the living body are not easily determinable, and are extremely difficult, or, in many cases, at present impossible to obtain in artificial cultivations. *Chemical specificity* has, however, already been established for three bacterial infections, namely, for diphtheria, tetanus, and anthrax. This was due first and chiefly to the work of Sidney Martin, whose results have been confirmed by numerous observers. It has been shown that the poisonous substances which can be obtained from cultures of these organisms are identical with those found in animals dead of the disease in question; and that both will, if injected into healthy animals, produce identical lesions.

Specific harmful substances produced by micro-organisms are usually spoken of as toxins. We may therefore say that a disease is proved to be specific when both its bacterial and its toxic specificity have been established. The conditions for specificity may accordingly be briefly stated as follows:

1. A particular micro-organism and no other must be invariably associated with the disease in question and with no other disease.

2. It must, when transferred to healthy susceptible animals, produce the disease in them, and be again discoverable in their tissues.

3. It must, if capable of cultivation artificially, produce the same disease when such a culture is inoculated in a susceptible animal, and must be again recoverable from the tissues of the animal inoculated.

4. The toxic substances obtained from artificial cultures, and from the tissues of diseased animals, must be in chemical and physiological agreement.

Since the harmful action of pathogenetic micro-organisms is exerted through the agency of chemical substances which they form, it is of the first importance to inquire into the nature of the chemical products of bacterial metabolism. Of these, unfortunately, only very little is known. The subject of bacterial chemistry is in its infancy, and is surrounded by peculiar difficulties, so that it is even more backward than the sister branches of pathological and physiological chemistry.

Among the numerous substances which may be obtained from cultures of bacteria I may mention briefly the following, namely, *gases* such as carbon dioxide, hydrogen sulphide, and ammonia; *acids* of the fatty and oxy-acid series, such as acetic, lactic, and butyric acids and their *amides* (*e.g.* leucin, etc.); bodies of the *aromatic series*, such as tyrosin and phenol; *indol;* pigments; ptomaines; toxalbumins; ferments and enzymes, and the products of fermentation. Many of these are common to a considerable number of bacteria, others are more or less specific or confined to a small group of organisms.

The gases, fatty and oxy-acids, aromatic substances and pigments, do not appear to be endowed with any specific harmful action.

The *ptomaines* were origininally thought to be the specific bacterial poisons. They are basic substances which were first investigated by Brieger, who compared them to the vegetable alkaloids, and considered them to be the specific toxins. They are, however, not alkaloids, but amines, and are produced especially in putrefactive processes. They are not invariably poisonous, nor do all pathogenetic bacteria produce such bodies. Frequently they are only present in bacterial cultures in quite small amounts, and they do not in general, when injected into animals, produce the specific lesions of the disease in question; accordingly they are no longer looked upon, even by Brieger, as specific toxic substances.

The immediate interest in these bodies was considerably diminished by the discovery by Hankin and Sidney Martin of *toxic albumoses* in bacterial cultures. Brieger and Fränkel also separated toxic albuminous bodies from diphtheria cultures, and applied the term *toxalbumins;* but Martin showed these bodies to be really albumoses. Working with definite solutions of albumins as culture media, he proved the formation of toxic albumoses in the case of anthrax, ulcerative endocarditis, diphtheria, and tetanus. This was especially important in view of the fact that the poison of snake-venom had been shown by Weir Mitchell to be a peptone—or more correctly, as was later proved, an albumose. And Martin's own experiments showed the active principle of abrin, a poison

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derived from jequirity seeds, to be also an albumose.

It has, however, been proved by Buchner and by Uschinsky that bacteria still can form their toxins when grown in culture media which contain no albuminous material; and Brieger and Boer have separated the toxins of diphtheria and tetanus from both cultures in a form in which they do not give any of the reactions of proteids at all. Hence it seems probable that the toxic substances are not albuminous in nature, but only intimately associated with these albumoses, with which they tend to be precipitated from their solutions. The chemical nature of the toxic substances themselves is quite unknown at present.

The fact that bacteria cultivated in solutions of albumins give rise to the formation of albumoses proves that they exert a digestive action on the nutrient media. They therefore act as organised ferments, and in the case of the diphtheria bacilli, at any rate, produce an enzyme which was found in diphtheritic membrane by Roux and Yersin, and by Sidney Martin. Martin, indeed, regards this enzyme as the true diphtheria toxin, which, by its action in the living body, gives rise to secondary toxic albumoses, and these in turn to the symptoms and the lesions of diphtheria.

But on this point it is at present impossible to make a definite statement, since all attempts to isolate such enzymes in a pure condition have hitherto entirely failed.

It has been usual to divide bacterial toxins into extra-cellular and intra-cellular varieties, according as they are or are not discharged by the bacteria into the surrounding medium. On the enzymic theory extra-cellular toxins would be such as are formed outside the body of the bacterium by the action of its secreted enzymes, not excreted ready formed by the bacteria. But, as we have seen, the existence of such extra-cellular enzymes is at any rate still uncertain; and the analogy of other organised ferments favours the view that even the extra-cellular toxins have been formed within the bacterial body and then excreted. Thus, in the case of alcoholic fermentation, alcohol has been shown to exist within the cells of the yeast during activity. It is therefore formed within the cell and afterwards excreted, and not produced by extra-cellular action as was formerly believed.

The toxins of diphtheria and tetanus are those best known as belonging to the class of extra-cellular toxins,—that is, such as are discharged by the bacteria into the surrounding fluid. In actual practice, however, the distinction between extra- and intra-cellular toxins should not be very sharply drawn, since in any culture of bacteria numbers of individuals are continually dying and become broken up, thus liberating any intra-cellular toxic substance which they may contain.

But whatever ultimately proves to be the method of formation, and the nature of the specific toxins, their action in susceptible animals, at any rate, is to produce the particular disease with which they are associated.

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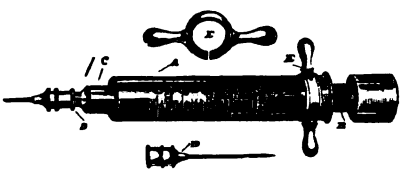

means of a perfect plug joint, and takes a needle (D) of the usual pattern. It will be seen that except during its passage through the needle the solution comes in contact with glass surfaces only, and the four essential parts of the syringe—barrel, piston, nozzle, and needle—are instantly detachable, and may be rendered perfectly aseptic. The required quantity of water should be drawn into the syringe, the nozzle removed, and a "Tabloid" hypodermic product dropped into the barrel. The nozzle having been carefully replaced, solution may be assisted by shaking. A detachable finger-grip can be used if desired. The syringe is issued in two sizes, min. 15 and min. 20.

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A CLINICAL LECTURE

ON THE

TREATMENT OF TYPHOID FEVER.

Delivered at Guy's Hospital, October 11th, 1902.

By W. HALE WHITE, M.D., F.R.C.P.,

Physician to the Hospital.

GENTLEMEN,—During the last fortnight I think we must have had some ten or a dozen cases of typhoid fever in the wards, so I thought we would continue what we were saying last time about this disease by telling you to-day some principles of the treatment of it. You must not imagine because there is no specific remedy for it, comparable, for instance, to diphtheria antitoxin, or quinine for malaria, that you cannot do much for sufferers from typhoid. So far from that being the case, there are very few diseases for which you can do more; and although there have been some ten or a dozen patients within the last fortnight that I have seen with you, I think I can safely say that if they had been treated as badly as they could have been, instead of being, as we believe they have been, properly treated, most of them would have died. So you see treatment has a good deal to do with the matter. You naturally must first of all put your patient to bed. You may say: Of course you do, because he is so weak that he ought not to do anything else. But unfortunately patients often try to do things when they are far too weak. For instance, the woman now in Mary Ward who was so bad when she came in, although ill, had gone about her work for more than a week. I remember a man who came in the hospital walked from Folkestone with typhoid fever on him, doing the journey by easy stages day by day. Because patients with typhoid fever often try to work it off, as they say, it is an important matter to put them to bed; whatever a man's engagements are, the moment you know he

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has typhoid fever he must go to bed absolutely. This is true of all diseases which are accompanied by considerable fever. I do not know that I can give you the whole explanation, but one is that if a man is expending a certain amount of energy by maintaining his temperature four or five degrees higher than in health, he has not so much energy left to walk about. And in the case of typhoid fever it seems to be particularly important to put the patient to bed. If there is anything wrong in the abdomen, and the patient has pyrexia, to bed he must go. Appendicitis, peritonitis, typhoid, dysentery, whatever it may be, this must be done. Sir William Jenner used to say that there was no disease in which it was more risky to move the patient than typhoid fever; and, indeed, it is difficult to think of one more risky. I have known patients moved with the greatest care from their own homes to the hospitals, and they have died soon after admission; and it seemed to be the moving which caused the death. So remember there is no disease about which it is more important that you should put the patient to bed, and at once, and that he should not be allowed to be moved. Having got him to bed, the next thing that occupies your attention is: What shall we feed him on? That is the kernel, I take it, of the treatment of typhoid fever. Now it is clear that the diet must be easily digested, easily absorbed, and by the time it reaches the ileum it must contain no large solid particles, for if it does there is a risk that they will break through the thin floor of the typhoid ulcers and set up peritonitis. I am sure many of you who are here think we are hypersensitive on the matter of food for typhoid fever patients, but, remember, we have to contend with visiting days. Early in my career I had the good fortune, or misfortune, to make a post-mortem on a patient with typhoid fever who had perforation, and I found watercress free in the peritoneal cavity. It is good that such experience should come early, because it stamped upon my mind for the rest of my career the importance of not letting any indigestible particles reach the ileum. And really, provided the food is easily digested and absorbed, and if solid particles which are in it disappear by the time it reaches the ileum, and it is non-irritating, you have fulfilled the conditions peculiar to typhoid fever. The reason why we are so strict here with milk is that in a large institution, where the friends are allowed to come in, you must be strict. Every Sister will tell you of the apples and such things turned out of the lockers of typhoid fever patients after visiting day; and if you allow them an egg beaten up in milk, which there is no reason why you should not, their friends say let them have an apple with it too. And as you cannot argue with the friends you must adopt a strict rule. But remember that in private practice, with the limitations which I shall tell you of directly, it does not matter so much what the diet is, but it does matter in a public institution. In private practice you can thicken the milk with any kind of infants' food, you can flavour it with chocolate, you can add an egg to it, or you can have an egg beaten up in it, or you can give the patient bread and milk, provided the bread is all crumb and grated. And in many cases, provided the food is easily digested and easily absorbed, and non-irritating and fluid by the time it reaches the intestine, there is no particular reason why you should keep to milk. However, we had better take the patients as we see them here, and we will keep to that aspect of the subject in this lecture. Milk is the mainstay of the patient's food, because it fulfils all the indications I gave you a moment ago; it is easily digested, it is easily absorbed, and if it is properly digested it is fluid by the time it reaches the ulcers, and it is non-irritating. Another advantage of it is that it is a very fluid diet, and all fever patients are thirsty. Another advantage is that no chewing is required, and these patients cannot secrete much saliva; no fever patients can. Another advantage is that, being fluid, it is easy to swallow, and many of these patients are so exhausted that they could hardly masticate their food.

Let us now try and see whether milk is enough; whether we want to add anything to the milk to make it an ideal food for typhoid fever patients. The physiology books tell you that a man in ordinary work requires about 3000 kilo. calories of energy per twenty-four hours, and he requires it in something like the following proportions:— From proteid, 125 grammes; from carbohydrates, 500 grammes; from fats, 50 grammes. When he is taking in about 3000 kilo. calories per day, that is the best proportion of proteid fats and carbohydrates to give. You will say that in thinking about the diet for typhoid fever, a man

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who has typhoid fever is doing very little work. That is true. It is true that the muscular work which you and I do is not accomplished in typhoid fever; that almost the only muscular work a typhoid fever patient is doing is that his heart is beating, his muscles of respiration are moving, and he is moving about a little in bed. There is also the energy necessary for the digestion and absorption of his food. But you will say he is raising his bodily temperature, and ought I not to be able to calculate how much food I ought to give him to do that? Practically you cannot. It is quite true that as an average man weighs about 70 kilos., and knowing that the specific heat of the body is about ·9 and that the temperature in typhoid fever is raised 2° C. or 3° C. as the case may be, we might conveniently be able to multiply it out and state how much food we should have to give him to keep his temperature at that height. But the impossibility of calculating accurately is two- or threefold. In the first place, it is very essential when you are thinking about any fever to remember that the temperature may go up for one of two reasons. The temperature will rise if you diminish the loss of heat or if you increase the production, other things being equal. And it is probable, for reasons I cannot go into now, that in different fevers the temperature is raised in different ways. In some fevers the temperature rises because the production is for the most part increased; in other fevers the temperature rises mostly because the loss of heat is diminished. Possibly in no fever is one of these causes operative solely. We did some work on this subject in the wards some years ago, when Dr. Salter was my house physician. We estimated the amount of sweat secreted and the surface temperature, and if these were increased we were able to conclude that the patient was losing an excess of heat from his skin; or if the sweat was slight in quantity and the surface temperature was low, the inference was that part of his rise of temperature was due to diminished loss of heat. In typhoid fever the temperature rises in considerable part because the patient diminishes his loss of heat, and that is what we should expect when we remember that there is no specific fever of anything like the duration of typhoid fever out of which the patient will so often come successfully in the struggle for life. There is a woman in Miriam Ward who is in her third relapse. She has been weeks in bed with a raised temperature, but she is not extremely wasted, possibly because the temperature has been maintained largely by diminution of the loss of heat. But be this as it may, even if we could settle the question as to how much of the raised temperature was due to diminished loss of heat, and how much to increased production, we still should require to know, in estimating how much energy was required to keep the man's temperature up some degrees, what was the temperature of the surrounding air. And the outcome of it all is that we cannot calculate exactly as a sum how much food we ought to give to compensate for the rise of temperature. But it seems pretty clear among clinical physicians that if a man of average weight has typhoid fever, with the average temperature of typhoid fever, you will be doing the best for him if you give him somewhere about 2000 calories of energy a day. You see, what we want is a thousand short of what is required for a man who is doing ordinary muscular work. Now let us look at milk diet in this connection and see whether it is at fault. We have seen that in health foodstuffs are required daily in the following ratio:—proteid, 12; carbohydrate, 50; fat, 5. In milk the relationship is this: proteid, 12; carbohydrate, 20; fat, 18. So the weak part of the milk is seen to be that it is extremely deficient in carbohydrate, and that it has got too much fat. Therefore, when you are giving your patient milk in typhoid fever, remember that the diet of milk contrasted with the proper diet for an adult is that it is very short of carbohydrate. And that is what every-day experience will tell you. Bread and milk is a far better diet than milk only. You know that in fever the chief tissue to suffer is proteid. See how the muscles of these patients waste; look at the large excretion of urea which people with fever have. And I will give you some figures when we come to another matter which will illustrate the same point. So in fever the patient is breaking down his proteid tissues excessively. You say: Why not give him plenty of proteid to make up for this? One or two objections strike us at once. In fever you cannot give enough proteid to attain nitrogenous equilibrium, and so you can never get matters quite square. And then you will remember from your physiology that fats and

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carbohydrates, and especially fats, will spare proteid. They are called proteid sparers. Surely it is far more economical, rather than giving a man with fever more proteid, to give him something which will spare the proteid which he has already got. Therefore you will readily see the indication is to give a fever patient an excess of carbohydrates or fats. Now, no fever patient will take a lot of fat; it is so nauseating; so you must give him a quantity of carbohydrate. And if you remember our figures you will see it is just carbohydrate that milk is deficient in. So when we are dieting typhoid patients we shall give them an ideal diet if we increase their carbohydrates in the milk. You have seen me do it many times. Sometimes we give the patients lactose. There are at the present moment two patients in John Ward who are taking lactose added to their milk. Or you may give them maltine, which is very good, and so, in fact, is any carbohydrate, *e. g.* treacle or honey. Maltine is handy, and it is a good thing to put into the milk. We come then to this—that for theoretical reasons we believe it is good to give our patients with typhoid fever milk, and to add to that a considerable quantity of carbohydrate. I will give you some very interesting figures to support my doctrine; I do not want you to take the statement from me without support. Here are some experiments done upon starving animals—it was necessary that the animals should be starving, for otherwise the results would not have been reliable.

A rabbit was losing on successive days 47, 50, 54 calories from its proteid tissue. At that point fever was induced by the inoculation of swine fever into the animal, and the daily loss from proteid for the next two days was 64 and 70 calories. You see how the breakdown from the proteid tissue increases. That is only one of many experiments which have been done by May and other observers, and shows that in fever the breakdown falls largely upon the proteids. Here is another very instructive case:

A rabbit which already had fever was taken. It was losing energy at the rate of 60 calories a day from its proteids, 171 from its fats. The fever continuing, sugar was put into the diet. At once the proteid-sparing effect is seen, for the breakdown from the proteid falls from 60 to 32, and the fat, too, was spared.

You remember that in one of Jules Verne's novels they have to break up the timber of the ship to stoke the fire so as to get round the world in eighty days. That is ruinous, and it must lead to the total destruction of the ship. But if they could have put some coal into the ship day by day to keep up the fire by that means no such catastrophe would occur. That is what you do to the fever patient by giving him carbohydrates. The temperature has to be maintained until the fever is over, and it is maintained at the expense of the carbohydrates which you put in rather than at the expense of the proteids of the body. We usually give three pints of milk a day in the hospital, which is equal to 1350 calories of energy a day, and the patient wants 2000. A dessertspoonful of maltine is worth 60 calories, and therefore, if we give a man half an ounce of maltine four times a day, we are giving him 480 calories from maltine in addition to 1350 from milk, or a total of 1830, which is about what he wants. So, the typhoid fever being of average severity, and the patient of average weight, you will probably supply him with the amount of energy that he requires if you give him three pints of milk a day and a tablespoonful of maltine four times a day. I think I have now shown you the principles on which you should go when you are dieting your fever patients. You will say: Are there any objections to the milk? Many people think it is very disagreeable, and it is very monotonous to take. So it is, but that monotony can be overcome very easily by flavouring the milk. I think that is best done by adding coffee or tea; a very little coffee or tea will flavour a pint of milk, and this obviates that disagreeable nauseating taste, and can do no harm to the patient. I admit a purely milk diet is very disagreeable to the patient after a time, and he gets to loathe it, but it is only in hospitals that he need keep to it. In private practice you can add biscuit powder, eggs, bread-crumbs, or infants' food to the milk. The most serious objection to the use of milk exclusively is, I think, that you get sometimes big curds in the stomach, and there is no doubt that they do produce severe diarrhœa, which is exhausting. No doctor has done his duty to a typhoid fever patient if, the nurse having directed his attention to considerable diarrhœa, he does not examine the stools. In a number of cases he will find the diarrhœa is due to the presence of curds

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of milk in the stools. But that is a difficulty easily got over. Peptonise the milk, and that cannot occur. So I think you can get over all the difficulties in connection with the giving of milk to your typhoid fever patients.

So much for the diet. The next point is, because it is a food, as to alcohol. Take the woman in Mary Ward and the woman in Miriam Ward. They have both been on alcohol, and the question constantly arises: Shall I or shall I not give my typhoid fever patient alcohol? What do you hope to do with it? A little alcohol helps digestion; there is no doubt of that. You know alcohol promotes sleep; you know it is a food; to some extent it is a diaphoretic. To some extent, also, it is an antipyretic. All those qualities are good. But you rarely give alcohol in typhoid fever for those reasons. You give it because of the pulse; and in health every one knows that alcohol increases the pulse-rate; it increases the force and frequency of the beating of the heart, and therefore you may say it is highly undesirable in typhoid fever. So it would be, but for some unknown reason when the patient has fever the alcohol frequently, instead of increasing the pulse-rate, slows it, and no one can tell, however large his experience is, whether alcohol will or will not benefit the patient until he has found by experience whether it slows the pulse.

Thus it comes to this—that if the patient is very severely ill and collapsed, and he is in danger of losing his life, do not attempt to stimulate the heart with alcohol, because you have to wait twelve hours before you can know whether you are doing any good. Give other drugs. But if the patients are not so bad as that, you may give alcohol, and you may or may not slow the pulse-rate. That was the line we went upon with the woman in Mary Ward. You have to remember with regard to alcohol it has this advantage—if it does slow the heart—over other drugs given for the same purpose, such as digitalis, it is a food. A tablespoonful of brandy is worth 100 calories, so a patient on three or four ounces of brandy is taking 800 calories in the way of food in alcohol. Another advantage of the alcohol is that it is a tissue sparer, as you see in people who take much of it, for often it causes them to become fat. It is burnt up in the body, and represents so much energy saved to the tissues, and especially the fatty tissues; and in that direction it is good. But to sum up, the whole pith of the question whether you shall give alcohol to a patient with typhoid fever is contained in your observation whether or not it diminishes the rate of the heart-beat.

You will remember the next thing we did for some of these patients, those who were bad enough, was to order them to be sponged with cold water. That raises a question in treatment: What shall we do in that direction in typhoid fever? I hope we shall get straight on that matter before we finish. The origin of the cold-water treatment of fever was rather interesting. There was a Dr. James Currie, who, in 1797, published a book which constitutes most interesting reading, 'Medical Reports on the Effects of Water, Cold and Warm, as a Remedy in Fever and other Diseases when applied to the surface of the body or used internally.' I rather like the length of the title, gentlemen. That is a book which I recommend you to read if you happen to come across a copy of it. Dr. Currie was on board ship with a Dr. Wright, who in those days was a distinguished physician in Edinburgh, and Dr. Wright one day said he had fever, and thereupon walked up on the deck of the ship and made the sailors throw buckets of water upon him; and Dr. Wright was benefited by the treatment. This so struck Currie that he treated his patients in that way afterwards, and that was the beginning of the cold-water treatment. You will say: Why does it do good? I do not know. We believe nowadays that the pyrexia of fevers is a protective mechanism; it is perhaps an attempt on the part of the body to sterilise itself. Then, you will say, why bring the temperature down? It is interesting in that connection that we thought typhoid fever a few minutes ago was a disease in which the temperature was up because the loss of heat was diminished, and cold water will increase the loss; but be that as it may, I do not know any theoretical reasons for sponging people who have typhoid fever that will bear very close examination. It is purely a practical reason. Are the patients, or are they not, better when it is done? They certainly often appear to be better. The reason, then, for applying cold in typhoid fever is that the patients do better for it. There are thousands of cases which have been published. The last collection is by Dr. Hare, of Brisbane, who has had charge of a very large fever hospital in Australia, and who has published his results. In 1828 cases not treated

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by baths the mortality was 14·8; out of 1902 treated by baths it was 7·5. That is striking enough to make you stop and think. But these statistics, I always maintain, are a very rough and ready argument. Although he has apparently diminished his mortality from 14·8 to 7·5 by bathing every one of 1902 consecutive cases, he has not shown that bathing may not kill some. Supposing, for the sake of argument, that the practice of bathing patients killed some and benefited others, but saved more than it killed, he would nevertheless get a lowered mortality. It appears to me that indiscriminate bathing is very haphazard sort of treatment. It is on a par with giving every patient with a murmur digitalis, quite independent of whether you thought the murmur was important or not. The right way to approach this subject is in every individual case to use your discretion, and see whether in that particular case you think that the application of cold will be beneficial or not. Often you find it is, but I only enter a protest against uniform bathing. I want you to use your judgment in each individual case. I agree that in a great epidemic you must have a hard and fast rule; in a hospital containing hundreds of typhoid fever patients it may be necessary to have standing orders that all patients whose temperature rises above a certain point are to be bathed, but generally you will be able to use your judgment about each particular case. You will notice whether the temperature seems to be affecting the patient; whether he is delirious, with a rapid pulse; whether he is comatose, losing flesh rapidly; and all those points will receive your attention before you decide about cold bathing or sponging. But usually you will find it beneficial to sponge a patient when he has reached somewhere about 103°. You will ask me: What is the good effect? I assure you that in a bad case the effect is most striking; the temperature falls, the pulse-rate falls, the coma passes off, and the delirium vanishes, the patient being obviously in every respect the better for the cold. I have known patients ask to be bathed. You will ask: Does it shorten the disease? No; it has been conclusively proved that it does not shorten the disease, and that it does not prevent relapses. Naturally you will say: Are there any contra-indications to it? Absolutely the only two contra-indications to this cold-water treatment are perforation, and profuse intestinal hæmorrhage. It has been shown over and over again that bronchitis is actually benefited by it if the bronchitis is due to typhoid fever.

The next point is: What shall we do? Shall we sponge or bathe? I think the experience at Guy's on that matter is good practical experience. With myself I have found that I cannot control the temperature except by bathing about once a year; that is to say, in one case a year which comes under my care I have to resort to bathing to bring the temperature down. I have never added up how many cases of typhoid fever one gets in the course of a year, but it must be considerable. You can often diminish the number of cold applications necessary, as I pointed out in the ward, by taking the temperature every two hours, because then you sponge before the temperature has got much mastery. You can use ice-cold water, and often you can improve matters by leaving a cold sheet over the patient, for then the evaporation from the cold sheet helps to keep the temperature down. If one day you are called to a patient with typhoid fever with a high temperature, and you go over the reasons we have talked over to-day, and you decide he requires cold applied; first of all tell the nurse to take the temperature every two hours and give him a cold sponging whenever it exceeds the point which you fix upon, say 103° or 102½°. If these methods fail you will have to give a cold bath. In summer-time the best plan is to take ordinary tap water; but in the winter-time it is necessary to put a little warm water to it. If you are going to give a cold bath it should certainly never be lower than 70°, and you can put your patient straight into a bath of that temperature. Take the temperature in the rectum, and when it falls to about 100° take the patient out, for it will continue to fall after he comes out. Then quickly carry him back to bed and throw a blanket loosely over him. Much more comfortable for the patient, but more difficult, especially in small houses, is to give a tepid bath gradually cooled. The temperature of this should be 10° or 15° below that of the patient. Therefore if his temperature is 105° you will put him in a bath of about 90°, and then you can, by adding cold water or ice to that, gradually cool it until it is down to 70°, or the patient's temperature has fallen to 100°. Then you can take the patient out and do the same as you did before.

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And now let us go back to the consideration of the cases which we have had in the wards. The patient in Mary Ward required enemata. If the diarrhœa is very profuse, and if it is not due to undigested milk, you may require to give enemata of starch and opium. But do not give the enemata too often. It is probable that the diarrhœa is a means of getting rid of harmful matters. Generally I do not think you will require to give any starch and opium unless the bowels are open at least four times a day; it will depend upon individual cases. The patient I notice on another occasion required a soap enema. Do remember, please, that people with typhoid fever are frequently constipated, in about one third of all the cases; and, as a rough rule, when you are dealing with a large epidemic and have to give general directions to the nurse, it adds to the comfort of the patient if you leave directions that if the bowels are not open for two consecutive days, then enemata should be given. I generally give olive oil in typhoid fever, but this particular patient had soap given to her.

We have already mentioned brandy and the indications for giving it, and I told you I would come to some drugs which may be given instead of it. There is no period of typhoid fever which is more difficult to manage than the end of the third week. It is most alarming to the friends. The patient has been ill a long while, and particularly then are the patients liable to die of exhaustion, and they may get most distressing attacks of cardiac failure. It is not infrequent for one to be sent for in a hurry, to be called up in the night to deal with such a matter, and the question is, what shall we do to ward off these attacks of collapse? There is no drug for this like digitalis. The woman in Miriam Ward is taking digitalis, and there are two prescriptions which I commonly use for it. As a rule, people give too little of it. I should give as much as 12 or 15 minims of tincture every three or four hours. You can always stop it if you find it is not doing good. Of course you ought to be seeing a patient like that twice a day, or oftener, and you can soon see how matters are going. So to begin with, 12 or 15 minims every four hours, and I think it helps them if you put in five grains of caffeine, and you must put in 2½ grains of sodium salicylate to dissolve the caffeine; and we add a few minims of tincture of nux vomica, 7 or 8, or 3 or 4 of the liquor strychninæ. With that medicine you can do an immense amount towards keeping the heart going towards the end of typhoid fever. Another favourite one is the infusion of digitalis, 2 drachms, with 3 drops of strong solution of ammonia in half an ounce of water. I am supposing, of course, that for this collapse you have found that alcohol is unsuitable, or that the collapse is too sudden for you to care to try alcohol for it. Very often musk is given, but there are many objections to that. In the first place many people do not believe in it, though I think I have seen it do good; and then, again, it is difficult to obtain, and it is only in the largest towns that chemists stock it. And it is very expensive. In private practice you may reckon it costs a guinea a day for musk if you are to give the patient enough musk to do any good.

The only further point which occurs to me in looking through the report of the woman in Mary Ward is that I see she had some beef-tea. I was not responsible for that, but it did not do her the slightest harm. You have to remember, with regard to beef-tea and the innumerable beef extracts, that they are not suitable for these cases. Think of what these things are. The extracts contain only what little soluble proteid there is, and all the extractives. The meat juices, of which there are many in the market, are more nutritious, because the juice is pressed out of the meat under pressure; but it contains chiefly extractives, and with regard to the little proteid it contains, we saw many reasons why it was not desirable to give proteids to patients with typhoid fever. The extractives are probably of little use to the typhoid patient. They are very nice; there is no doubt about that—good beef-tea and good meat extract is very palatable; they both help digestion and stimulate it, but that is all you can say for them; there is very little energy in them. If you want to give these patients a stimulant, give them something like alcohol, which contains a certain amount of energy and acts as a stimulus at the same time.

Mr. BUTLIN, in the first paragraph of his lecture in the issue of this Journal of December 10th, over-stated the mortality of removal of the upper jaw. The statistics he took are those of the German from König's Klinik. The English and American mortality is much smaller, and does not amount to more than about 13 or 14 per cent. But it cannot be said that the English cases were complete removal of the upper jaw. Some of them were and some of them were not. Fuller information is to be found in the second edition of Mr. Butlin's book on 'The Operative Surgery of Malignant Disease,' pp. 127 to 130.

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SOME DETAILS IN THE REMOVAL OF ADENOIDS.

Remarks at a Practical Demonstration, London Throat Hospital, November 5, 1902.

By E. FURNISS POTTER, M.D., M.R.C.P.

ALTHOUGH—as you see the operation performed in hospital—it is apparently a simple affair, I venture to think that the remarks I have to offer, and the suggestions I shall make, will be of some value in drawing your attention to several essentially practical points of detail, which are of considerable importance for the satisfactory and efficient performance of the operation.

I presume that you have seen the operation frequently, and also that by digital examination you have made yourselves familiar with the feel of the parts in the naso-pharynx, such as the posterior border of the septum, the Eustachian cartilages, and the "adenoids" themselves.

The first point to which I would draw your attention is the choice of the anæsthetic. In my own mind I have no doubt whatever that for all but very young children a combination of ether and nitrous oxide gas is by far the most satisfactory. I know that some men of eminence still prefer chloroform, but, having had a considerable experience of both, I most unhesitatingly advise "gas and ether" as *the* anæsthetic for this operation. More than this, I would warn you to beware of chloroform in these cases. I do this because I am so convinced of the (I might almost say absolute) safety of the "gas and ether" if properly given; whereas with chloroform, no matter how carefully administered, there is always (at any rate it has been so in my experience) a certain proportion of cases which give rise to anxiety. With ether I contend that, if the breath-way be kept clear, there should be no need for anxiety whatever, *i. e.* on account of the anæsthetic.

As to the anæsthetist, it is important that he should not only be experienced in the administration of anæsthetics, but that he should be familiar with adenoid cases. He should know how much ether to give so as to induce anæsthesia to last, say, for at least three or four minutes after the removal of the inhaler, in order to allow the operator plenty of time to proceed leisurely in his work, and that he may not have to complete the operation when the patient has partially come round and begun to struggle. I consider there is nothing more unsatisfactory, more annoying, or more to be deprecated, than attempting to remove adenoids in a patient insufficiently anæsthetised.

At the London Throat Hospital the anæsthetist administers ether to such a degree as in his judgment will give an anæsthesia of about three minutes. It is sometimes impossible to gauge this quite accurately, so that he has a Junker's chloroform inhaler within reach, in case the patient shows signs of "coming round" too soon, or if from one reason or another there be any delay and the time necessary for the completion of the operation be longer than usual. The Junker is useful to have in case of need, but, as a matter of fact, it is rarely necessary for it to be called into requisition, as the patient can generally be "charged up," so to speak, with ether to such an extent that the duration of anæsthesia will be amply sufficient for the performance of the operation.

The position of the patient is a matter on which opinions differ. Some prefer the semi-recumbent position, others that the patient should be lying on his side. I always operate with the patient lying on his back, and the head slightly hanging over the edge of the table, *i. e.* with the occipital protuberance just overhanging the edge. In this position the vault is the lowest part of the pharynx, the blood flows through the nose and mouth, and the likelihood of any finding its way into the larynx is minimised.

The holding of the gag is a matter worthy of mention. This appears to be simple enough, but, as a matter of fact, it is of considerable importance, and I would advise you to always have some one on whom you can rely to keep the gag firmly in position. From what I have said you will see that you are in great measure dependent—for the smooth running and complete success of the operation—on your anæsthetist and gag holder. I lay some stress on this point, and speak from personal experience, having on one occasion been much inconvenienced by an assistant who was incapable of placing the gag in position, and when I had done this for him was unable to retain it there.

There is usually free bleeding, necessitating frequent sponging, which is done, as a rule, either by

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the anæsthetist or the gag holder. Occasionally the anæsthetist both holds the gag and attends to the sponging; this, however, I consider too much of a demand upon one pair of hands, but it has to be done sometimes. As to sponging, I would utter a word of caution against sponging with too much haste and force and too little care, which often results in considerable contusion of the soft palate and neighbouring parts. Sponge firmly and deliberately, but gently, taking care that the sponge is properly fixed in the holder, and that the end of the latter is well covered.

A point worthy of mention is, that the anæsthetist should avoid standing between the patient and the light; attention to this certainly adds to the comfort of the operator.

Choice of Instruments.—A number of different instruments have been devised and extolled by various operators, but there are only two that need be mentioned—these are the forceps (of various shapes and makes) and the curette (Gottstein's). Opinion is divided as to which is the better instrument. Personally I use both. In certain cases the curette is highly satisfactory, as when there is one central clump of growth which can be removed with a single sweep of the instrument. In using the curette, care has to be taken to avoid the lateral parts of the pharynx, for fear of injuring the cartilages of the Eustachian tubes; so that, as I have said before, the curette is excellent for removing a central clump, but is not so suitable where the growths are scattered, and situated at the side of the pharynx, and in close proximity to the Eustachian cartilages. Here the forceps is much preferable. In some cases I have found that even after repeated trial the curette would not catch the adenoids, but would slip over them. There is also another fault of the curette against which it is well to be on the look-out. The clump of growth may have been cut through, but has failed to be completely severed, and is left hanging low down in the pharynx by a thin ribbon of mucous membrane. It is possible that this, owing to the bleeding, may escape notice at the time of operation, unless you very carefully look out for it, and when you come to examine the patient's throat the next day you may be annoyed by seeing a tag of growth just visible below the edge of the soft palate, and held there by a tiny shred of mucous membrane. It is surprising with what energy these tags contract adhesion to the pharyngeal wall, so that, if not removed, in a few days they may become firmly adherent.

This, of course, should not happen if you are alive to the possibility, and its occurrence may be minimised by having the blade of the curette kept very sharp. Another reason for its occurrence is, that in the sweeping movement given in using the instrument, the blade is not brought sufficiently low down on the pharyngeal wall before withdrawal, and so fails to effect complete severance.

Of course the tag is easily removed, but there may be some trouble with a nervous patient, and in the case of a child an anæsthetic may be required, which, for obvious reasons, one would very much prefer to avoid.

It is said that this trouble can be almost completely avoided by using a modification of Gottstein's curette, which consists in the addition of prongs to the instrument, on which the growths become impaled and are brought out on the blade. There is usually more hæmorrhage with the curette than with the forceps, but not to such an extent as to be counted an argument against the use of the former.

With the forceps the operation takes more time, but I consider it on the whole to be more certain and reliable than the curette. It is, I think, almost unnecessary to utter a caution against seizing the uvula, or taking a piece out of the posterior border of the septum, though these things have been done. If the growths are tough and resistent when seized, I think it a good plan to twist them off rather than pull forcibly on them, as, though not very likely, there is a case on record where the greater part of the mucous membrane of the pharyngeal wall was stripped off by this means.* Of course, in the majority of cases you will have to remove the tonsils also. This is so simple a procedure as not to call for any remark, except that I would advise you to be guided by the sense of touch rather than by that of sight in slipping the ring of the tonsillotome over the tonsil.

The hæmorrhage may be, and frequently is, brisk, but, as a rule, very quickly ceases, and rarely is such as to cause anxiety or to call for special treatment. It is generally from the stumps of the

* 'Diseases of the Nose,' Greville MacDonald, p. 259.

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tonsils. If unusually profuse, I find the readiest and most efficient means of stopping it is to apply a sponge wrung out of hot water (from 115° to 120°), and keep it pressed against the bleeding surface for a few seconds, exerting pressure with the forefinger on the sponge, and the thumb through the neck. This generally suffices.

Perhaps it may be thought that the points I have drawn attention to are of such minor importance as to hardly merit the time spent. However, I am taking it for granted that I am addressing those whose experience in this operation is limited; this being so, I am sure that if the hints I have given be remembered, considerable difficulty will be avoided and the thoroughness of the operation will be more certainly assured.

TUBERCULOUS MENINGITIS IN ADULTS OCCURRING AS A TERMINAL EVENT IN CHRONIC PULMONARY TUBERCULOSIS.

By S. VERE PEARSON, M.B.Cantab., M.R.C.P.,

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Tuberculous meningitis occurring as a terminal event in the chronic pulmonary tuberculosis of adults presents many points of interest which are not so well recognised as the commoner varieties of the disease. This form differs somewhat from the type of tuberculous meningitis seen in babies; there are special difficulties in its diagnosis; and its relationship to general tuberculosis is of some theoretical and practical importance. The following conclusions arrived at are arranged under these three heads, taken in the above order. They are founded upon a study of some thirty cases which have happened during the last few years at St. George's Hospital and at the Brompton Hospital for Consumption, several of which have come under my own observation.

Tuberculous meningitis in adults occurring as a terminal complication in chronic pulmonary tuberculosis differs from the disease seen in infancy in the following respects:

1. A prodromal stage is more commonly ill-defined or absent, and the course of the disease is shorter.

2. Definite signs of meningitis, such as vomiting, retraction of the abdomen, headache, irregular or very slow pulse, paralyses and convulsions, are much more commonly absent or much later in appearance than is the case in babies. Head-retraction is particularly rare, and optic neuritis comparatively rare, in adults.

3. Mental symptoms are prominent in the adult.

For these reasons the diagnosis of tuberculous meningitis in such cases is even more complicated and difficult than is usually the case with this disease. This form of meningitis in adults has often to be diagnosed more from a series of somewhat minor and ambiguous symptoms than from any obvious sign of the disease. In discussing the diagnosis it will be convenient to divide the patients into three classes:—those in whom the signs of chronic pulmonary tuberculosis are (1) well recognised and advanced; (2) recognised but not advanced; and (3) absent or indefinite.

There is probably a difference in many cases in the part played by the meningitis as a cause of death between the patients of the first class and the patients of the other two. Some patients with advanced pulmonary tuberculosis may have meningitis without this being the cause of death; they die from exhaustion and toxic poisoning as a result of the pulmonary disease. They die with meningitis, but not of it. The exhaustion and toxæmia produced by the chronic disease so lower the resistance of the system towards the end that an active extension and dissemination of the tuberculous process throughout the body occurs. This active deposition of tubercle is a terminal, but not necessarily the terminating event of the exhausting chronic disease. Hence sometimes it is found that patients dying of advanced chronic pulmonary tuberculosis have a fairly extensive meningitis without having shown any well-defined clinical manifestations of it. Now all patients nearing the end of a chronic, exhausting, and wasting disease are liable to sink into a state of excessive helplessness; they become semi-conscious, irrational and irritable, dirty and perverse; and in addition sometimes there is vomiting, incontinence of fæces and urine; and perhaps the patient will lie curled up, turned away from the

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light, and the temperature will be raised, or become higher and less intermittent. It is into such a state as this that the patient with tuberculous meningitis sinks. And supposing, as very frequently happens, no optic neuritis, severe headache, convulsions, paralyses, or other definite signs of meningitis arise, when, of course, the diagnosis becomes clear, it is difficult to differentiate between the patient dying with meningitis and the partially demented patient undergoing gradual dissolution without meningitis. There are, nevertheless, the following minor points of difference to aid in the differentiation between the two conditions:

1. In the patient undergoing gradual dissolution, as a rule the period of time covering this stage of partial dementia is shorter than the time during which similar symptoms due to meningitis are present.

2. The partial dementia of approaching dissolution is constant, and gets progressively worse; the patient will not answer questions, and seldom speaks; whereas the irrational mental condition in the case of the patient with meningitis varies from time to time; the patient, at one time sullen and silent, will at other times talk either sensibly, somewhat irrationally, or altogether in a rambling way.

3. Death is imminent in the one case, but with meningitis death may arrive somewhat unexpectedly.

4. The action of the sphincters, when they are affected, differs slightly in the two cases. Meningitis is more likely to be associated with retention, the other condition with incontinence of urine. In uncomplicated chronic pulmonary tuberculosis, if one sphincter is affected, generally both are affected; but the irregularity in the action of the bladder in the case of meningitis is less likely to be associated with irregularity in the action of the anal sphincters. It usually happens that a patient in such a state about to die of chronic pulmonary tuberculosis, if loss of control occurs, suffers from incontinence of both urine and fæces during the last forty-eight to seventy-two hours of life; whereas in meningitis some irregularity in the action of the sphincters will occur for a longer period.

5. Meningitis is more likely to be associated with constipation, and the impending dissolution with diarrhœa.

6. With regard to the taking of food, and its retention in the stomach, in both conditions frequently food is refused, and vomiting may occur; but, as a rule, when food is refused, that refusal is persistent in one case, whereas with meningitis there is great variability and capriciousness of appetite.

The points of difference between the tuberculous meningitis in this class of case and that meningitis which occurs in the two classes next to be considered are as follows:

Meningitis in I:	*Meningitis in II and III:*
The chronic pulmonary disease is advanced and the general condition of the patient bad.	The chronic disease is not advanced, and may be very early, and the general nutrition of the patient is often good.
Death is sometimes due more to the chronic wasting disease than to the meningitis or accompanying general tuberculosis.	Death is due directly to the meningitis, or to the general tuberculosis which is associated with it.
The symptoms of the meningitis are more indefinite.	The symptoms of meningitis are less indefinite.
Many of the symptoms referable to the nervous system are explicable by the theory of septic absorption and nervous exhaustion.	Symptoms referable to the nervous system are less easily explained in such a way, and are more suggestive of meningeal inflammation.
Active delirium and headache are less common.	Active delirium and headache are more common.
The pulse is rapid and feeble.	The pulse may be full, and is often not rapid.

In considering the second class of case, *i. e.* those cases in which the signs of chronic pulmonary disease are well marked but not advanced in degree and extent, there is little to be said in the matter of diagnosis which is not included either in the foregoing or in the points which are considered below. The diagnosis is generally rendered comparatively easy by the recognition of the pulmonary lesion. I will mention, however, the following case which was recently brought to my notice as an illustration of the kind of difficulty which may sometimes arise. The patient was a male who had well-marked signs of chronic pulmonary tuberculosis of three years' duration, but not advanced. He developed peculiar mental symptoms, which lasted, up to his death, for many months. At one stage of this changed mental state a diagnosis of general paralysis of the insane was suggested by his practitioner, and there were certain resemblances to this disease. However, this case did not run at all the usual course of general paralysis of the insane, either as to the length of

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time or as to symptoms. There was a possibility of the brain condition being due to cerebral tumour, chronic tuberculous deposit in all probability. Unfortunately, a post-mortem examination was not obtainable, and the diagnosis finally made, *i. e.* chronic pulmonary tuberculosis and tuberculous meningitis running rather a more chronic course than usual, could not be confirmed.

We now come to the consideration of the diagnosis in the third group, where the terminal meningitis occurs in a patient with chronic pulmonary tuberculosis in whom the signs of the primary affection are indefinite or absent. If there have been any symptoms of the primary disease, then the discovery of the existence of such symptoms may afford a valuable clue in arriving at a correct diagnosis of the existing condition. But frequently no such helpful clue is afforded, the first symptoms of disease arising with the onset of the fatal complication. The terminal meningitis in these cases may give rise to a sudden onset of symptoms, or the onset may be quite gradual. It seems that where the onset is sudden the course tends to be shorter than in those cases where the onset is gradual. A typical example of a case belonging to this third class, those without definite signs of the pulmonary lesion, was recently in St. George's Hospital under the care of Dr. Penrose, to whom I am indebted for permission to quote it here.

A clerk æt. 20 was admitted with a temperature of 101° complaining of loss of appetite, great weakness, and some headache. For nearly three months he had had occasional headache, attacks of dyspepsia, slight cough, and some general weakness; he was sufficiently well, however, three weeks before his death to go for a long march with his regiment of volunteers. But at the end of the march he fainted, and three days later took to his bed and became a little queer in his head. On admission, ten days before death, he was fairly well nourished; his cerebration was very slow; he took a long time to answer a question, and his answers were not always rational. Moreover he would not always carry out a command reasonably. Later, sometimes he would not answer questions at all. He tried to get out of bed once or twice, and he did not like being disturbed. There was never any vomiting, paralyses, rigidities, or loss of sensation. Loss of control of the sphincters occurred during the last ten days; but there was no optic neuritis, the fundi being examined by several observers on more than one occasion. The tendon reflexes were normal, and no photophobia occurred. There was neither cough nor obvious dyspnœa or cyanosis until a few hours before death, when both these symptoms arose, and at the same time moist râles appeared all over the lungs. Previously the only abnormal physical signs in the lungs were very slight impairment of resonance to percussion in the left infraclavicular fossa, with one or two doubtful clicking râles after cough in the same region. Owing to the perverse mental condition of the patient an accurate and exact investigation of the apices was rather difficult, and the signs found were very indefinite.

Such a case, presenting none of the ordinary definite signs of meningitis, except that he usually lay curled up and rather drowsy, suggested the possibility of several diseases; and I have quoted it to illustrate some of the difficulties of diagnosis which may arise under such circumstances. In considering the differential diagnosis of tuberculous meningitis as it occurs in adults, associated, as it so often is, with symptoms of this kind, several diseases suggest themselves, *e. g.*—

Differential Diagnosis from Enteric Fever.—In enteric fever the same low and feeble state of body, the same lack of interest in the world around, with a weak state of mind, delirium, and fever, occur. In the absence of typhoid eruption, Widal's reaction, or some other more or less definite sign of typhoid, the diagnosis must depend on the following points:

1. The temperature in enteric tends to be higher than is the case in tuberculous meningitis. With regard to the type of temperature, the occurrence of remittency or intermittency is commoner in meningitis. But this cannot be relied upon, because sometimes, for a time at least, the temperature in tuberculous meningitis is scarcely remittent at all; and on the other hand, cases of enteric occasionally have a very markedly remittent, or even an intermittent, temperature.

2. Interference with the action of the organic reflexes is not common in enteric, whereas retention of urine is fairly frequent, and incontinence of fæces and urine is not infrequently present in tuberculous meningitis.

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3. Some reliance can be placed upon the type of mental change. The enteric patient is likely to have chiefly delirium with lucid intervals, whereas the patient with tuberculous meningitis, though the mental symptoms may vary from time to time, appears to be partially affected mentally all the time. Moreover there is often in tuberculous meningitis a characteristic irritability of the patient not seen in other fevers.

4. Dr. Judson Bury * refers to Sir William Jenner's observation, namely, that the headache ceases when delirium begins in typhoid fever, but in brain disease the two may co-exist, as a point of importance in considering the differential diagnosis between these two diseases.

Differential Diagnosis from Insanity, Hysteria, and Delirium Tremens.—As a general rule the irregular mental phenomena which are liable to occur in these cases are associated, if not with definite symptoms of organic disease, at all events with much feebleness or other bodily derangement, which should give the clue to the existence of disease of organic origin. In this connection particular importance must be paid to undue complaint of head pain or the occurrence of vomiting. But occasionally, for a time at all events, they present such peculiar mental phenomena, with so few signs of organic disease, that insanity is naturally thought of, and the patient is sometimes sent to an asylum before the true nature of the case is discovered. This result might sometimes be obviated by remembering not to disregard the rule of taking the temperature in every mental case. Dr. Clouston† may be aptly quoted in connection with this. He says, "Never think any examination complete without taking the temperature. Many patients labouring under the delirium of fevers and inflammations would have been saved from being sent to asylums had this been done." These patients, too, do not present a mental change conforming to any well-recognised type of mental disorder, and they are very prone to pass suddenly into a dazed condition, and thence into coma. The rapidly fatal issue should always suggest the correct diagnosis in these cases if it has not already been arrived at before the fatal result ensues. Reference has already been made to general paralysis of the insane. Many of the mental symptoms which arise, again, are not unlike certain manifestations of hysteria; therefore tuberculous meningitis must be borne in mind, in some cases suddenly starting curious pseudo-hysterical phenomena. Delirium tremens, also, is a disease from which tuberculous meningitis may possibly have to be differentiated, as is mentioned by Oppenheim * and Barlow.†

Differential Diagnosis from (a) Cerebral Tumour, and (b) Other Forms of Meningitis.—In the absence of the recognition of the presence of a chronic tuberculous lesion the diagnosis must depend upon the more rapid course of meningitis, and, on the other hand, on steady progressive intensity in the symptoms of cerebral tumour. Further, optic neuritis is more likely to occur, and to be more profound, in tumour than in meningitis. Sir William Gowers lays stress upon the "early occurrence of limb symptoms and the gradual loss of power" as indicating tumour rather than tuberculous meningitis; but the diagnosis between these two is especially difficult when the intra-cranial tumour gives no localising symptoms, or when it occurs in a patient with chronic pulmonary tuberculosis. Reference has already been made to a case where this differential diagnosis could not be made with certainty. The differentiation of tuberculous from other forms of meningitis is dependent upon the general principles laid down in the text-books. The most difficult cases of all are the rare conditions occasionally met with, such as a primary localised tuberculous meningo-encephalitis, which later becomes generalised. It must be remembered also that very rarely tuberculous meningitis runs a very chronic course. Oppenheim ‡ refers to a form with intermissions and long duration, and mentions a case associated with hydrocephalus which lasted over a year. Osler,§ too, says that there are "instances in which the acute process is associated with chronic meningeal tuberculosis; cases which may for

* "Tuberculous Meningitis in Adults," 'Internat. Clin.,' Philadelphia, 1897, i, p. 202.

† Clouston's 'Mental Diseases,' 5th edit.

* Oppenheim's 'Lehrbuch der Nervenkrankheiten,' 1902, p. 680.

† Clifford Allbutt's 'System of Medicine,' vol. vii, p. 466.

‡ Ibid.

§ Osler's 'Principles and Practice of Medicine,' 3rd edit., p. 279.

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months present the clinical picture of brain tumour."

General Remarks on Points Helpful in Diagnosis.—Little is to be learnt from a study of the kind of patient in whom tuberculous meningitis arises during the course of a chronic pulmonary tuberculosis. Of the cases I have collected twenty were between 21 and 35 years of age, one was 63, the two youngest were 18 and 15, and the age of the remaining seven varied from 35 to 54; but this roughly accords with the usual death-rate at various ages from chronic pulmonary tuberculosis. Males predominated amongst these (twenty-one males and nine females). With regard to the nature of the pulmonary lesion and the general condition of the patient, these varied so much that no clear indication could be made out as to the kind of patient in these respects in whom the complication was likely to arise. Although rather commoner in advanced disease, and not infrequently associated with ulceration of the larynx and intestines, tuberculous meningitis seems to arise in a patient with a lesion of small extent and a well-nourished body almost equally easily as in a case with advanced disease and great emaciation. The only point noticeable was this—that in the majority, whether the chronic disease was advanced or not, it was usually active, and sometimes in the history predisposing depressing circumstances could be discovered leading up to a recent activity in a previously quiescent lesion.

There is nothing typical about the behaviour of the temperature in the tuberculous meningitis of adults. The temperature is always raised at some period of the disease. The temperature is generally as follows:—With the earliest onset of symptoms of meningitis the temperature rises, usually one and a half to two degrees. Then for a few days it will tend to stay at that point, with but slight remissions. Later, with the advent of severer symptoms, it will become very irregular and variable, sometimes sinking to normal, sometimes becoming violently intermittent, and sometimes, especially if death is imminent, rising higher than before.

By a study of the pulse little generally is learnt pointing to meningitis, unless irregularity or marked slowing occur, but these only occasionally occur as early signs of meningitis. So also with respect to the reflexes; and, so far as I have investigated it, I have not found Kernig's sign an early or a reliable one. Its absence certainly does not prove, as is now recognised, the absence of meningitis, though the presence of this sign when clear certainly weighs much in favour of the disease.

In one only of the cases that I have studied was lumbar puncture performed. In that case turbid fluid was withdrawn. This, nowadays, is a very helpful adjunct to the means of diagnosis. Osler refers to the evidence obtained on lumbar puncture as a point upon which special stress is to be laid. He says, "The fluid withdrawn is usually turbid, and in it, on centrifugalising, the bacilli may be discovered."

The Diagnosis of the Extent and Nature of the Lesion.—All the thirty cases I collected had well-marked lesions. But in morbid anatomy I can discover no definite variation in adults from the various forms of tuberculous meningitis met with in infancy and childhood. The same morbid lesions occur, and no factor governing the variety of the lesions which occur was discovered. Nor could I make out any definite relationship between the variety or extent of the lesion and the variety or degree of severity of the symptoms. For example, I thought that, since pressure symptoms were somewhat less common and definite in adults than in babies, excessive fluid in the ventricles or subarachnoid spaces would be less common in adults than in babies. But, so far as I can tell from thirty cases in adults, such is not the case. I was unable either to confirm or to refute Sir T. Barlow's statement that meningitis of the convexity is more rapidly fatal, and presents earlier convulsions.

The Connection with General Tuberculosis.—Tuberculous meningitis as a terminal event in the chronic pulmonary tuberculosis of adults is almost invariably associated with general tuberculosis. In all except two of the thirty cases I have collected miliary tubercles were found elsewhere than in the meninges; generally scattered in the lungs, with a few in the kidneys, and frequently, in addition to this, in other organs, such as the spleen, liver, peritoneum, and pleuræ. In those rare cases in which fresh scattered miliary tubercles are not found elsewhere than in the meninges, it is fairly certain that the tubercle bacilli have spread by the blood, and in all

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probability miliary tubercles exist elsewhere, but are too minute to be observed. An occasional exception to this occurs when tuberculous meningitis has spread directly from chronic tuberculous deposits in the brain. In the two cases, where discrete miliary tubercles were not found scattered in other organs, this was the state of affairs, for in both of them tuberculous tumours were present in the brain.

The question as to whether general tuberculosis is so commonly associated with tuberculous meningitis as the latter is with general tuberculosis is more doubtful. Probably meningitis is nearly always present in some degree in cases of general tuberculosis. Although general tuberculosis without meningitis is a common post-mortem appearance, this is no evidence that tuberculous meningitis has not occurred without producing visible deposits. It is doubtful, however, whether a small degree of meningitis, not demonstrable to the naked eye at the autopsy, can in adults give rise to the well-recognised symptoms of the disease. In infancy I feel convinced that this is so occasionally, and I am inclined to think that in adults, too, the same thing may occur. The following case is of interest in this connection :—An emaciated patient, æt. 63, was recently admitted into St. George's Hospital for retention of urine and great weakness, feverishness, and pains in the stomach. He had chronic tuberculous lesions in the peritoneum and in both lungs. He had retention of urine throughout, and later incontinence of fæces. He was peculiar in his behaviour, frequently refusing food, and talking irrationally; later he became delirious, and finally semi-comatose, and died a fortnight after his admission. He never had any paralyses, fits, vomiting, or headache. There were râles and rhonchi all over both lungs. At the autopsy, miliary tubercles were found in the lungs, peritoneum, and kidneys, but no sign of meningitis was discovered by the naked eye. It is easier to explain such a case as this by the supposition that many of the symptoms were due to meningeal involvement than by supposing that the interference of the action of the sphincters and other signs suggestive of meningitis were due to other causes, though it is possible that the very weak bodily state of the patient, associated with the general tuberculosis and with the local tuberculosis in the abdomen, would cause such interference and symptoms. Spinal tuberculous meningitis, in association with cranial tuberculous meningitis, is, according to Osler, very rare; though, according to Still and Seitz, it is common, and was found in fifteen out of twenty-two and in twelve out of twenty cases respectively examined by them. It was not eliminated in the above case, for the spinal cord was not examined at the autopsy. If, then, this supposition is correct with regard to the great prevalence of meningitis in cases of general tuberculosis, the diagnosis of meningitis in a certain number of cases is removed from the clinical field into the field of fine pathological research.

Probably minor degrees of tuberculous meningitis are much commoner than is generally supposed. And I would go further than this, for I believe that a minor degree of general tuberculosis is a much commoner event than it is usually thought to be. I do not wish it to be understood that I think general tuberculosis a very common event in chronic pulmonary tuberculosis. But it is possible, I think, that a certain number of the febrile disturbances which occur in the course of chronic pulmonary tuberculosis are due to the presence of tubercle bacilli in the blood. The majority of these disturbances are without doubt due either to fresh activity and extension of the disease locally, or to some minor complication. But as a rare event, when no evidence of such a cause for the constitutional disturbance can be discovered, often when the resisting powers of the patient to the invasion of tubercle have from one cause or another become lessened, may it not be that the tubercle bacilli getting into the blood attempt to set up general tuberculosis? A constitutional disturbance is produced, but at the same time the properties possessed by the blood and the fixed cells of the body, properties resisting and antagonistic to the deposition of tubercle, are brought out by the extra exciting stimulus; they act more vigorously, and fatal general tuberculosis is averted. The constitutional disturbance is, amongst other things, nature's call for rest, and helps in postponing the fatal issue. Probably the presence of tubercle bacilli in the blood is not necessarily followed by the formation of miliary tubercles. It is difficult to bring forward direct evidence in favour of such a view; but there is a certain amount of indirect evidence. The recovery from general tuberculosis, with the formation of a

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few scattered miliary tubercles in many organs, is an excessively rare event. Many are sceptical about such a recovery, and yet most physicians have come across some case with good clinical evidence of general tuberculosis which has recovered, and, in addition, have seen on the post-mortem table a few calcified miliary tubercles (or, at all events, an appearance indistinguishable from this) scattered in the organs of a patient dying from some totally different disease, or sometimes from tuberculosis. If all the evidence in favour of such recovery could be brought together, I feel sure that the occurrence of occasional recovery from general tuberculosis would be universally accepted as an incontestable fact. Moreover we have such suggestive facts as the following:—The ease with which chronic pulmonary tuberculosis becomes healed in a number of cases. The old healed lesion is often found in patients who have never been troubled by symptoms. Tubercles are frequently present in the liver in cases of chronic pulmonary tuberculosis (M. Simmonds). Tubercle bacilli are of common occurrence in the urine of patients with chronic pulmonary tuberculosis (Foulerton *). And the occasional presence of healed miliary tubercles in the choroid is another suggestive fact (Carpenter and Stephenson †).

In this connection it is of interest to refer to Dr. H. Head's ‡ observations on the mental changes associated with chronic pulmonary tuberculosis. He believes that the occasional mental changes which occur in pulmonary tuberculosis are excited by referred visceral pains. He describes, amongst other things, hallucinations of vision, and moods, such as the "sense of ill-being." No doubt grave visceral disease may so lower the stability of the mind that some mental aberrations of this kind arise, and perhaps "visceral reflected pain and its accompanying superficial tenderness" are direct causes of these aberrations. But the factors which govern our mental perceptions and sensations are so obscure that it is difficult to say definitely what are the exact causes governing the mental changes in disease. If an organic basis is sought, possibly the visions, moods, and suspicions arising in patients suffering from chronic pulmonary tuberculosis are dependent on a slight degree of generalised tuberculosis with some intra-cranial involvement—meningeal or in the cortical end-arteries.

Conclusions.—In conclusion, I should like to emphasise the following points:

1. Tuberculous meningitis of this kind does not as a rule present clinically the well-marked characteristics of the disease seen in childhood. The duration of symptoms is shorter; the onset may be sudden or insidious, but in either case easily recognisable signs of meningitis are frequently absent, either throughout, or until the last few hours.

2. Retention of urine occurring in a patient with chronic pulmonary tuberculosis, whether the disease be advanced or only slight, should always suggest tuberculous meningitis where no local cause for the retention can be found, for it is often one of the earliest symptoms of this disease.

3. Curious mental or pseudo-hysterical phenomena with a raised temperature should always arouse suspicions of tuberculous meningitis, whether signs of a chronic tuberculous lesion are found or not.

4. In patients dying of chronic pulmonary tuberculosis who have exhibited slight mental symptoms shortly before death tuberculous meningitis is sometimes found post mortem when unsuspected and when no other more definite symptoms of the complication have been present.

5. It is almost invariably associated with general tuberculosis.

These remarks, I believe, would apply equally to the tuberculous meningitis of adults secondary to chronic tuberculous lesions other than pulmonary. Dr. Mainwaring Holt* has published a case occurring in a woman of 25, secondary to chronic tuberculous disease of the hip, in which he lays emphasis on "the extreme difficulty of making an early diagnosis, and the rapidity of the disease." And there is one case of tuberculous meningitis, in the records of St. George's Hospital, occurring in a woman of 46, which was secondary to chronic glandular tuberculosis and unassociated with any chronic pulmonary lesion, in whom the features of the disease were very similar to those just described. There was retention of urine, short course (three days), semi-coma, and death, without any convulsions, vomiting, or paralyses.

Undoubtedly chronic pulmonary tuberculosis is by far the commonest lesion to which the tuberculous meningitis of adults is secondary.

* 'Lancet,' 1901, vol. ii, p. 550.

† 'Lancet,' 1901, July 20th; and 'Reports Soc. for Study of Disease in Children,' 1901.

‡ "Goulstonian Lectures," 'Brain,' vol. xxiv, part 3, 1901.

* 'Brit. Med. Journ.,' 1897, vol. i, p. 1281.

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THE DIAGNOSIS AND TREATMENT OF INFECTIVE THROAT DISEASE.

An Address delivered before the Wimbledon Medical Society.

By F. FOORD CAIGER, M.D., F.R.C.P.

MR. PRESIDENT AND GENTLEMEN,—I think one is justified in asserting that the only forms of sore throat of an undoubtedly infective character with which the medical profession in this country is liable to be confronted are those associated with either diphtheria, scarlet fever, or influenza, though cases of tonsillitis are not infrequently met with which we have reason to believe are of a septic nature. With your permission I propose to devote the time at my disposal this evening to a brief allusion to certain practical points in respect to their diagnosis and treatment which experience has shown to be of value. And in so doing, should I find myself in conflict with other views which have obtained a general or wide-spread acceptation, I shall not hesitate to indicate the particulars in which I believe them to be erroneous.

It might be objected at the outset that the foregoing enumeration is defective in that it makes no provisions for the inclusion of the sore throat which is a recognised manifestation of a common cold, than which few disorders are more infective. This, no doubt, is perfectly true, but it is very doubtful indeed whether such infective catarrh is ever characterised by a sore throat alone without very obvious involvement of the neighbouring mucous surfaces; and when considering the diagnosis of the various forms of infective throat illness, it has no more claim for inclusion than a disease such as measles, in which more or less injection of the fauces is invariably present.

For the purpose of discussing their diagnosis, the various forms of faucial inflammation, or, in other words, "sore throat," may be definitely separated into two groups, according as to whether

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or not they are attended with any visible exudation. To make a really satisfactory examination of the faucial structures often requires a good deal of knack on the part of the examiner, so greatly does the control over that rebellious organ, the tongue, vary in the case of different patients; as also does their inclination to retch on the slightest attempt to depress the tongue with a spoon or spatula. This is especially true when dealing with young children. And since with them the moment at which the act of retching occurs often provides the best opportunity for obtaining a complete view of the faucial region, it is well to get the light focussed on the throat before attempting to introduce the spatula, in order not to miss the opportunity for inspection which the act of retching offers. It is in this way usually possible to obtain a momentary view of the mucous surface covering the base of the tongue, the anterior surface of the epiglottis, and the lateral aspect of the pharynx which is normally obscured by the more prominent tonsils. This latter situation is not infrequently found to be the seat of a patch of exudation which is very likely to be overlooked if the examination has been cursorily made, or the illumination inadequate.

I would here add a note of warning against an error which is sometimes committed, viz. of mistaking for an exudation an accumulation of Schneiderian mucus which has descended through the posterior nares and temporarily ensheathed the posterior pharyngeal wall. It can, however, be usually distinguished by its smooth, slippery appearance, its yellowish tint, and the fact that it can be dislodged with the end of a dry camel's-hair brush.

Having, then, come to the conclusion, after a careful inspection of the throat, that no exudation is present, we are justified in assuming that the affection with which we are confronted is more likely to be scarlatinal or influenzal than diphtheritic. I use the term "diphtheritic" as synonymous with true diphtheria, and not as a descriptive term to denote a merely exudative appearance resembling that disease, as I regard the use of the term in this sense as not only unscientific, but as tending to confusion. Or, of course, the case may be one of simple tonsillitis, which may be regarded provisionally as of a non-infective character. If the patient be a young child the chances are greatly in favour of the affection being scarlatinal, since the large proportion of sore throats in children of tender years are either scarlatinal or diphtheritic in their nature. Tonsillitis, catarrhal, septic, or influenzal, is a relatively uncommon disorder in childhood. In the case of scarlatina the diagnosis is not likely to present much difficulty if the characteristic eruption be present, especially if its punctiform or finely papular element be well developed; but in instances in which the rash is very transient or ill-defined, of a patchy or purely erythematous character, and particularly if it be suppressed or absent altogether, the diagnosis may not be by any means equally apparent. The distinctions which are of value in the differentiation between scarlet fever and diphtheria I will refer to later when dealing with the diagnosis of the latter disease. The indications on which we must rely in endeavouring to form an opinion as to whether a doubtful sore throat on which neither exudation nor rash can be detected is of scarlatinal origin, or a case of simple tonsillitis, are yielded, partly by the appearance of the throat, and partly from a consideration of other symptoms and circumstances connected with the attack.

Firstly, as to the throat. The strictly scarlatinal factor in the throat of scarlet fever is a vivid red injection of the whole of the fauces, palate, and buccal mucous membrane. There may be much, or there may be little, if any, swelling or ulceration of the tonsils. Its amount, though in the septic form of the disease of considerable importance from the point of prognosis, is, as it were, purely a matter of accident in respect to the diagnosis of scarlet fever. Some of the most toxic and rapidly fatal attacks are attended with no greater swelling of the faucial structures than is the rule in the typical benign or simple form of the disease, but the mucous surface in each case is the seat of a vivid red injection. This diffuse redness of the palate and buccal surface, though apparent at the commencement of the attack in the large majority of cases, fades rapidly, and in mild attacks by the end of twenty-four or forty-eight hours may only be apparent on the tonsils and along the free border of the velum and uvula, which then come to present a relatively deep red and tumid appearance. Other indications, which are of greater value than the appearance of

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the throat in the diagnosis of these doubtful attacks of scarlet fever, are the presence of nausea, if not actual vomiting at the outset, and undue rapidity of the pulse at an early stage. This latter symptom in adults may not be very apparent, but with children the commencement of scarlet fever is usually characterised by a rapidity of the pulse out of proportion to the height of the temperature. A pulse-rate of 140 to 160 is not at all uncommon in the first twenty-four hours of the scarlatinal attack in a child. Early cleaning of the tongue is evidenced by commencing redness of the tip and edges. This is usually noticeable by the end of the second day, and extending centripetally, causes the organ to become clean and raw-looking by the third or fourth day of the attack. In cases in which the throat affection has been very mild this cleaning up of the tongue may not be very complete, and the organ may never attain the typical raw or strawberry-like appearance. The degree of peeling which the tongue shows will usually be proportionate to the intensity of the antecedent injection of the mucous lining of the mouth and throat. But even in such mild cases, although the peeling process may by no means be complete, a careful inspection of the tongue will often reveal a scarlatinal character. The association of an inflamed throat with a tongue possessing this peculiar appearance is a symptom of the highest importance in the diagnosis of scarlatina, for there is no other form of sore throat which is accompanied by a clean tongue, much less a raw one.

Another sign of great value in these anomalous attacks is the appearance of pain with tenderness and more or less stiffness of the joints of the metacarpus, fingers, or wrists during the latter half of the first week. The time of appearance of this rheumatic complication is most constant, viz. the fourth to the seventh day of illness. It may be but slightly marked, and the patient regard it as hardly worth mentioning. It is, however, one of the most distinctive indications of the scarlatinal nature of the attack, and is often useful in confirming a suspicion we may have previously entertained. Should the patient have been exposed some days before developing a sore throat to the infection of a case of undoubted scarlet fever, the inference of course could hardly be ignored. The fact of the patient having previously suffered from scarlet fever, should the interval have been of some years' duration, is not so much opposed to the diagnosis of scarlet fever as might be supposed. It is perfectly true that most persons only contract the disease once in their lives; but, on the other hand, these anomalous cases of scarlet fever in which no eruption is detectable are most often seen in infants, adults, and those who have had a previous attack. During the convalescent period of the illness, confirmatory evidence of scarlet fever might be yielded by the appearance of desquamation, or some recognised complication of the disease, such as inflammation of the middle ear, glands in the neck, or the kidneys. So much, then, for the chief points of distinction between a scarlatinal and a non-specific faucial inflammation, whether mild or severe in degree, or catarrhal or septic in its nature.

Now I think it will be within the experience of most medical men who have been for some years engaged in general practice that, during the course of an epidemic of that protean disease influenza, cases of tonsillitis, often of such severity as to deserve the appellation of quinsy, are apt to be unusually frequent. In some instances the patient is obviously suffering from influenza at the time; in others the throat affection in one individual is closely associated with cases of influenza in the family, or amongst those with whom he has been brought into contact. The frequency with which such cases of tonsillitis present themselves varies considerably in different epidemics, and at different periods of the same epidemic. In some outbreaks tonsillitis will perhaps constitute the most common and obvious manifestation of the influenzal virus, attacks of this nature occurring in regular series; at other times they may be relatively infrequent, and occur only as isolated instances, interspersed here and there amongst cases of influenza of the common catarrhal variety. There is nothing in the local characters of the throat affection to distinguish it from an ordinary tonsillitis. The general constitutional disturbance is usually more severe; the headache, often referred to the back of the eyeballs, and the muscular and arthritic pains, are commonly far more intense and persistent. An excessive repugnance to nourishment in any form, vomiting and profuse perspirations, frequently of an intermittent character, are often present, and the pyrexia is likely to be unduly protracted. These symptoms, coupled with an un-

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usual degree of prostration and tedious convalescence, will commonly serve to indicate the influenzal nature of the attack, apart from its possible connection with other cases. In my experience influenzal tonsillitis is rarely met with in children, but at times when the disease is prevalent such possibilities should be remembered, if only in view of the liability to cardiac dilatation, apart from the desirability of enforcing isolation.

Let us now consider the diagnosis of those cases of sore throat in which a definite exudation can be detected on some part of the faucial structures. In the majority of instances the case will prove to be one of diphtheria; indeed, the presence of exudation in itself may be regarded as *primâ facie* evidence in favour of the diphtheritic nature of the attack; but by no means necessarily so, for an exudation practically indistinguishable in its naked-eye appearance from that of diphtheria is occasionally present on the tonsil both in scarlet fever and in tonsillitis, and this fact is not perhaps so generally recognised as the frequency of its occurrence would appear to warrant.

Now, of those cases of scarlet fever which are attended with an exudation resembling diphtheria, it may be stated generally that they are almost invariably severe attacks, and the characteristic symptoms of scarlet fever are usually so pronounced that the presence of that disease can hardly be overlooked. I refer especially to the puncto-erythematous rash, the strawberry tongue, the initial vomiting, and the frequent presence of rheumatic pains in the wrists and finger-joints towards the end of the week. Diphtheria, it is true, is sometimes attended with a transient erythematous eruption, but no punctation is to be detected in it, and the flush is usually limited to the trunk and arms. The difficulty in such instances is usually to decide whether the exudation is purely scarlatinal, or whether, as is sometimes the case, it is an attack of scarlatina complicated with co-existent diphtheria. The exudation of scarlet fever is practically always limited to the tonsils; it is of a cheesy consistence, and is easily broken up with a camel's-hair brush, and tends to disappear spontaneously in a day or two. If it be due to co-existent diphtheria, on the other hand, the exudation is usually thicker, tough, and gelatinous or fibrous-looking, incapable of removal except by force and with laceration of the subjacent tissue, and it separates *en masse* after an interval of several days. In a case of uncomplicated diphtheria, of course, the exudate may be thin, friable, and easily detached, but in these concurrent attacks of scarlet fever and diphtheria the condition is usually grave, and the exudation as a rule presents the characters I have mentioned. As far as my experience goes, it is more common for a case of diphtheria attended with an erythematous flush to be mistaken for scarlet fever, than for a case of scarlet fever with exudation to be mistaken for diphtheria.

But it is mainly in connection with follicular tonsillitis, and that form of sore throat which is usually regarded as of a septic character, in which the tonsils are liable to present flakes or small particles of dirty-looking exudate, that the differentiation of faucial diphtheria is likely to present the greatest difficulty. Indeed, the differentiation may be impossible apart from a bacteriological examination of the secretions. It is now customary to regard the presence or absence of the Klebs-Loeffler bacillus as sufficient to determine the diagnosis. Although in the large majority of cases this relation holds good, a more extended observation has shown that the test in actual practice—that is, as usually carried out—is not by any means so reliable an indication as was originally believed; and this, for two reasons. In the first place, not only do we occasionally come across a typical, and even severe, or fatal attack with clinical appearances distinctive of diphtheria, in which careful and repeated examination of the throat and secretions fails to reveal the appearance of a single diphtheria bacillus, but characteristic and even virulent bacilli are occasionally found in the throat secretion of perfectly healthy persons, especially if they have been in contact with cases of diphtheria. Indeed, one of the most typical and virulent cultures of diphtheria bacillus which I ever remember to have seen was obtained from the throat of one of my medical colleagues who was at the time, and subsequently, in his normal health, with no suspicion even of a sore throat. And the same thing may sometimes be demonstrated in respect to the nurses who are working in diphtheria wards. In a series of 140 cases of scarlet fever in which the throat secretion was examined by Professor Sims Woodhead, a large portion were found to contain diphtheria bacilli.

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And Ranke, of Munich, in his published results, states that more than fifty-two per cent. of his scarlatinal throats contained diphtheria bacilli. Then, again, in the rhinorrhœa or nasal discharge which is so common in scarlet fever, especially in young children, the presence of the diphtheria bacillus is extremely frequent. Todd, of the London Fever Hospital, has shown that in a few instances these diphtheria bacilli, in cases of scarlet fever, were really virulent; so that the presence of the diphtheria bacillus, in cases which on clinical grounds we assume to be scarlet fever, is no indication of their not being so. Whatever bearing this may have on their infectivity, one would certainly not be justified in asserting that these persons were suffering from diphtheria. Although a negative bacteriological examination cannot unfortunately be regarded as quite conclusive, for reasons to which I will again refer, a strong presumption is made out in favour of the cases not being diphtheria if confirmed by a subsequent negative examination, especially if the observer be an expert.

The second, and to my mind even stronger argument against the infallibility of the bacteriological test is the wide difference of opinion which exists amongst eminent bacteriologists as to what is and what is not a diphtheria bacillus. A bacillus which is regarded by one observer as a true bacillus which has undergone a temporary attenuation, is held by another equally experienced observer as a *pseudo*-bacillus, or one which is of an entirely distinct species, and incapable, under any circumstances, of acquiring virulent pathogenic properties. To the first observer the case is one of diphtheria, potentially at any rate; to the other it is not a case of diphtheria at all. Until the limits of normal variation, both in respect to form and virulence, can be agreed upon, no practical differentiation is possible, and as long as an appeal to the results of injection into the lower animals is necessary for a decision, the test must remain of but partial value in practice.

Having now briefly alluded to the disabilities which attach to the bacteriological test in the diagnosis of diphtheria, one may summarise its authority in actual practice as follows:—A positive bacteriological examination of a throat presenting definite follicular exudation, or from the larynx in a case of croup, even without visible faucial exudation, may be taken as sufficient evidence of diphtheria; but the finding of what appear to be diphtheria bacilli in a throat which presents no visible exudation cannot be held to be distinctive unless confirmed by inoculation into animals. A negative examination of material taken from a throat in which no exudation can be detected may be held to exclude diphtheria if the examination has been made on at least two, preferably three, occasions; but in the event of the examination proving negative in a case in which the appearance of the throat is distinctly in favour of diphtheria, it is wiser to ignore the bacteriological evidence altogether. It cannot be too strongly insisted that the diagnosis of diphtheria should never rest on the bacteriological evidence alone, but rather on the clinical appearance of the case supplemented by the result of bacteriological examination. Having regard to the fact that an interval of a week or so is required for putting in practice the test of animal inoculation, and that a negative examination, to be at all satisfactory, requires confirmation, it is obvious that the customary report by telegram at the expiration of twenty-four hours from the time at which the material is taken from the throat can hardly be expected to carry conviction, except in cases in which the clinical indications are in themselves fairly distinctive, and these certainly constitute the minority amongst those which are submitted to the verdict of the bacteriologist.

Now, the procedure required for obtaining material from the throat for the purpose of bacteriological examination—in other words, the taking of a swab—is so familiar to all that I feel some apology is due for even referring to it; yet, on the other hand, it so often occurs in practice that a negative result is contradicted by a subsequent examination, that I may, perhaps, be excused if I add a word of caution as to its proper performance. It should be remembered that a negative result can never be held to be absolutely conclusive. It simply implies failure to find on the part of the observer, not necessarily that diphtheria bacilli are really absent, apart from the doubt which not infrequently exists as to their specific identity. The failure may occur at either one of three stages in the experiment. *Firstly*, bacilli, though present in the throat, may escape attachment to the swab, either in consequence of their being scanty in

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number or partial in their distribution. *Secondly*, though present on the swab, bacilli may not grow on the culture, either through failure of inoculation, through impaired vitality of the organisms themselves, or in consequence of there being something unfavourable in the condition of the culture medium. And *thirdly*, bacilli, though actually present on the stained cover-slip preparation, if they are but few in number may escape detection when subjected to microscopical examination.

Either of these fallacies may occur, and in view of such possibilities the following precations should always be observed:—(1) Never take the swab immediately after the use of any antiseptic throat application. (2) Examine the throat with a good light, note the situation of any apparent exudation, and press the swab firmly against it, rotating it at the same time two or three times, in order to ensure its being thoroughly impregnated with secretion. (3) Rub the surface of the culture medium firmly with the swab, moving it up and down, and at the same time rotating it, in order to ensure the whole circumference of the swab being brought into contact with the blood-serum. (4) Avoid using a culture tube which has been kept too long, as when the surface of the blood-serum is very dry it is unfavourable to microbial growth.

A due regard to these particulars will ensure more trustworthy results, and the importance of this can hardly be exaggerated in view of the important *rôle* which the bacteriologist plays in the diagnosis of diphtheria at the present day. Our confidence in the bacteriologist is in part attributable to our knowledge of the ætiology of the disease, and in part, perhaps, to the weakness of human nature; for who does not prefer to shift a measure of his responsibilities, at any rate, on to other shoulders, and usually less assailable? Information of considerable value, because of its early availability, can often be derived by making an immediate cover-glass examination of the material on the swab. Few practitioners, I take it, are likely to be possessed of a bacteriological incubator as an item of their equipment, but to anyone who owns a microscope fitted with a $\frac{1}{12}$ inch objective, and can lay claim to even a limited acquaintance with the staining and examination of bacteria, such examination is readily available. For the purpose of identification of diphtheria bacilli I can strongly recommend the use of Neisser's double stain. Second only to the crucial test of animal inoculation, and the characters which are revealed by cultivation, both of which procedures require some time for their elucidation, I regard Neisser's stain as the most reliable criterion of the identity of diphtheria bacilli which we at present possess. Its advantages are twofold. It frequently reveals the presence of distinctive diphtheria bacilli in a cover-glass preparation made direct from the swab, and it is therefore immediately available; and further, it stains diphtheria bacilli in such an obvious and distinctive fashion that their nature may be readily distinguished by an observer who is not an expert bacteriologist. In an investigation into the diagnostic value of Neisser's stain which I carried out in conjunction with Messrs. Beaton and Pakes last year (the results of which were brought before the Pathological Society, and subsequently published in the 'British Medical Journal,' September 21st, 1901) it was shown that of cases subsequently proved to be diphtheria a positive result was obtained from an immediate examination of the swab by Neisser's method of staining in 37·5 per cent. of cases. Its value, therefore, as an immediate and readily available test of the presence of diphtheria, and its consequent importance, both in respect to diagnosis and treatment, will be apparent In cases in which the Neisser stain shows no evidence of diphtheria on the swab, a subsequent examination of the culture should never be omitted. For the information of anyone who is not familiar with Neisser's double stain, the actual composition of the two solutions is to be found in Muir and Ritchie's 'Manual of Bacteriology.'

Having now appraised the bacteriological test at its proper value in the diagnosis of diphtheria, let us see what clinical indications are likely to be available for the differentiation between follicular tonsillitis and diphtheria. The task of distinguishing between these two affections is rendered none the less difficult owing to the fact that in some cases of diphtheria the exudation commences at the very points which are most characteristic of follicular tonsillitis, namely, at the mouths of the tonsillar crypts in the form of spots or little patches of exudate, which at a later stage enlarge and ultimately become confluent, so as to form a continuous pellicle covering the surface of the tonsil;

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but at an earlier stage of the attack the appearance in each case is very similar. The clinical features on which some reliance may be placed are that in follicular tonsillitis the spots rarely coalesce, and the exudation never spreads to adjacent parts, but remains limited to the tonsils. Both tonsils are usually affected very much to the same degree, and the same thing holds good for the swelling of the subjacent glands, while in diphtheria it is, perhaps, more common for one side to be involved more than the other, and for the gland swelling to be proportionately greater. In follicular tonsillitis albuminuria is far less common, but the temperature is usually higher,—in fact, the general assertion is justified that the constitutional disturbance in follicular tonsillitis is likely to be more pronounced than in a case of diphtheria of sufficient mildness to be mistaken for it. It should be remembered, too, that in a child the attack is more likely *primâ facie* to be one of diphtheria than tonsillitis, because in childhood the latter disease is relatively uncommon. Laryngeal implication never supervenes, nor does any subsequent paralytic affection, whether of the palate, accommodation, or skeletal muscles, so distinctive of diphtheria. The foregoing distinction practically holds good for the differentiation of diphtheria from a mild scarlatinal throat, and there is one other point of distinction which is still more pronounced in respect to scarlatina than in the case of simple tonsillitis. The colour of the fauces, palate, and uvula in scarlatina is usually of a somewhat dusky red, and the mucous membrane often presents a relatively dry or even sticky appearance; whereas in diphtheria the fauces are more likely to be of a pale pinkish tint rather than a deep red, and the faucial surface bathed in thin glairy mucus which gives to it a slippery rather than a sticky appearance. In many instances all of the above distinctions may not be apparent, but when pronounced, such indications are not devoid of significance.

Lastly, in respect to that form of tonsillitis which it is customary to regard as of a septic nature the swelling of the parts is apt to be considerable, and the surface of the tonsils presents a dirty greyish appearance, which is commonly of necrotic origin, and though simulating diphtheritic exudation, the material is not separable as a distinct pellicle. Nevertheless, a superficial ulceration is usually apparent at a later stage. There is commonly a good deal of nasal discharge, and considerable infiltration of the glands and cellular tissue in the submaxillary region. The relation of such cases to diphtheria is not very clear. They tend to arise under conditions which are favourable to the development of diphtheria. But that they are not diphtheritic is attested by the absence of diphtheritic bacilli in the secretions. Various micro-organisms are present in the throat, some of which are doubtless putrefactive in their function. Both streptococci and staphylococci are found in most instances, but the organism which is, perhaps, most intimately concerned in the morbid process—at any rate it is commonly present in large numbers in the necrotic patches, sometimes in nearly pure culture—is a large diplococcus which is not known to possess any distinctive pathogenic attributes. The general aspect of the case is more like severe septic scarlet fever than anything else, but the resemblance to diphtheria at an earlier stage is equally pronounced. These attacks are apparently neither of scarlatinal nor diphtheritic origin, but they occasionally arise in children who have recently suffered from one or the other of these diseases.

Treatment.—It is well recognised that in all cases of throat inflammation local treatment is of considerable importance, especially in such as are of an infective character. Local treatment, to be effective, should have three aims. It should ensure, or at any rate it should be directed, towards the cleansing of the surfaces from any inflammatory or offensive secretions; it should inhibit as far as possible the multiplication of micro-organisms, whether specific, septic, or putrefactive; and it should conduce to a healthier state of tissue, and thus promote repair. For this purpose there is no doubt that an antiseptic and astringent lotion is best adapted, and it should be used either in the form of a gargle, or by swabbing, painting, spraying, or syringing the throat every few hours. Among the various solutions which I would recommend I think the best is an acid solution of chlorate of potash containing free chlorine. It is a very old remedy, and is very valuable for cleansing septic throats. A solution of chinosol, 1 in 600, is a good alternative, and easy to dispense, as it is made up into small tablets, each of sixteen grains, and all you have to do is to dissolve one in a pint of water to obtain a solution of the required

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strength. Another alternative is a half per cent. solution of formalin, but it is somewhat irritating, and has rather a tendency to catch the breath if a patient is at all inclined to be breathless. A much milder application, and one which is quite suitable and sufficient for throats which are not of a dirty or exudative character, is a simple solution of boric acid. In these four solutions you have got a selection which will be found sufficient for all ordinary purposes. The surface of the throat, and if necessary the naso-pharynx, should be irrigated and cleansed by means of a syringe, say every two, three, or four hours, according to the severity of the case. Of course, where the patient will assist you, the oftener you cleanse the fauces the better; but when the patient, as in the case of a young child, strenuously resists treatment, it is better not to be too insistent; but if the cleansing be efficiently done, even young children, who at first may strongly object to the performance, will themselves soon recognise the value of it, and even lend a hand to their own discomfiture. I have brought a syringe with me which I can very strongly recommend for the above purpose. It is an ordinary 4-oz. ball syringe, which is usually known as a "rubber enema bottle." The nozzle is made of vulcanite, in order to withstand decalcification by an acid solution; nor can a child bite it, as would be possible were the nozzle made of bone. The best way of tackling a child, and the same thing applies in the case of an older patient, is to stand on the right-hand side of the patient, who sits up in bed with a basin on his lap for receiving the fluid as it runs out of the mouth: take the syringe in the right hand, and use your left for steadying the patient's head. Introduce the syringe at the side of the mouth, pass it backwards over the tongue, empty the syringe with a sharp squeeze, and then withdraw it. By this means you accomplish your object very much quicker and more efficiently than by half squeezing the ball, and so letting the fluid trickle, as it were, out of the nozzle. Another point as to which some caution must be observed is not to push the syringe too far back into the throat, and to guard against this, the nozzle is provided with a shield about an inch and a half from its end. I have known of cases in which the palate has been damaged by contact with the point of the syringe when the patient has been a struggling child. After the syringe has been emptied some five or six times you will have exhausted a pint or so of the solution, and that will be sufficient. In cases in which there is much rhinorrhœa, and the nares are more or less blocked up with it, it is desirable to syringe them also, but you will not be justified in using the same amount of force in doing this as in cleansing the fauces and throat; otherwise you will run some chance of transmitting a dangerous amount of pressure into the middle ear. I maintain very strongly that there is no method of treating a septic throat which is to be compared with this in point of efficiency. No amount of gargling, spraying, or swabbing will be equally effective, because neither of these methods is capable of washing out or effectually cleansing the back of the fauces or naso-pharynx.

(*To be concluded.*)

Sciatica and Atropine.—The treatment of sciatica is a difficult matter, because the origin of the disease is so difficult to reach. The writer has been treating this condition for some time with large subcutaneous injections of atropine. Although these large doses determine the usual symptoms of intoxication more or less distinctly, they appear to have a decidedly beneficial result upon the pain. This observer takes 0·06 gramme of atropine in 100 drops of water, and of this injects three drops at night, about ten centimetres below the buttock of the affected limb. In about twenty minutes all the physiological effects of the alkaloid in question appear—namely, acceleration of the pulse to 120, dilatation of the pupil, more or less vertigo, and intense thirst. If these signs disappear in less than ten hours he supposes the reason to be that the dose was insufficient. The dose is therefore increased by one drop until these symptoms of intoxication continue for twenty-four hours. A second injection must not be given, however, until all the symptoms of the first dose disappear; consequently only one injection a day is required. Twenty-eight patients treated in this manner, including the writer, give a record of twenty-two definite cures. S. C. Felt (*Semaine Médicale*, No. 36, 1902).—*Monthly Cyclopædia*, November, 1902.

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WITH DR. E. W. AINLEY WALKER AT GUY'S HOSPITAL.

GENERAL PATHOLOGY OF INFECTION—II.

At the end of the last lecture we were considering the disease-producing products of bacteria.

Now, different species of animals vary in their *susceptibility* to any given disease, and of the members of the same species some are more susceptible than others. Animals whose susceptibility to a given disease is greater than the average for their species are said to be *predisposed* to the disease in question. Similarly certain Species may be predisposed to a particular disease, as in the case of guinea-pigs and tuberculosis.

Predisposition in a species is obviously an hereditary character, and must be dependent on peculiarities of the cell-processes proper to the animals concerned. An individual predisposition may be similarly intrinsic and dependent on cell-properties either hereditary or acquired, or it may be extrinsic and accidental—the result of circumstances which act injuriously upon the normal tissue-processes, lowering the resistance of the body.

Predisposed animals tend to become infected and to succumb to the disease, and thus fail to propagate, while the less susceptible escape. The natural action of disease is, therefore, to improve the physical standard of the species by the survival of the fittest only. In the case of man, the natural tendency to physical improvement along this line is, to some extent, continually thwarted by the efforts of the physician, whose duty it is to preserve the unfit in life so long as possible.

After recovery from an attack of an infective illness, susceptibility is found to have disappeared, and remains absent for a longer or shorter period. The insusceptibility acquired may last throughout a lifetime, as is usual in small-pox, for example, or may speedily be lost, as in pneumonia and influenza.

This observation that one attack of an infective malady can to a greater or less extent protect from subsequent infection by the same disease is probably one of the oldest in medicine, and its practical application is of some considerable antiquity. During the plague at Athens it was recommended by Thucidydes that the care of those attacked should be the duty of the convalescents, since they would not thereby acquire infection.

Again, the custom of variolation against small-pox was already old in 1717, when Lady Montagu first introduced it into Western Europe from Constantinople, where it was then a usual procedure. And long before that date, in India and in China, it had been the custom to seek a similar protection by means of insufflations of the powdered material of dried small-pox scabs.

Similarly, we are told that certain tribes on the West Coast of Africa have been for many generations, and still are, accustomed to expose their children to the contagion of framboesia, which runs a more favourable course in early life. And even among European mothers a similar course of conduct is not unknown with regard to the infection of rubeola.

These practices, though venerable, were purely empirical, and it is only within the last few years that we have begun to have some knowledge of the intimate processes concerned. These processes and the conditions which affect them we must now examine.

If a number of animals of different species be subjected to the action of the same infective agent, some only are found to be susceptible to its influence, others invariably remain indifferent. And of the members of a susceptible species exposed to an infection by an epidemic of a given disease some only will acquire the disease, while others, under apparently identical conditions, escape attack. Among the latter are included those which, by recovery from a previous attack, were rendered insusceptible. But there remain a number which have not thus been rendered insusceptible, but which can none the less successfully resist infection. And yet of these a certain portion may in a later epidemic prove susceptible, and even highly so.

The condition special to such individuals or species as show themselves either continuously or on occasion insusceptible to a specific virus is called immunity to that virus; it is the converse of predisposition.

This condition is only relative, not absolute. It is the function of two variables, the resistance of the organism and the virulence of the infective agent; accordingly it varies with their variations. Hence, by increasing the virulence of a particular

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bacterium, we may enable it to be successful in attack upon an animal which could previously resist it, or by sufficiently attenuating the same property we may deprive the bacterium of its power to invade a previously susceptible animal, which is thus found to be immune to the attenuated virus. Again, the virulence of the infecting agent remaining constant, we can increase the resistance of the animal—without reference to specific treatment—by increasing in any manner its general health and well-being, or, on the other hand, can lower it by any procedure which will lower the general level of the normal metabolic processes. Very numerous experiments have been made in this direction. Thus, Canalis and Morpurgo found that in inanition pigeons become susceptible to anthrax; Fermi and Salsano showed that at abnormally high temperatures chickens are easily attacked by tubercle; and Pasteur and Wagner observed that when subjected to low temperatures the same animals became less immune to anthrax. Similarly, immunity may be diminished by anæmia, deprivation of water, dyscrasias of the blood, fatigue, nerve-poisons, extirpation of organs, general diseases, such as diabetes, bad food, and many other causes.

Immunity, then, is a purely relative condition. It is therefore easy to understand why an individual who has successfully escaped one epidemic of disease may, on a subsequent occasion, show himself susceptible to the same disease, either because his own resistance has become lowered, or because the virulence of the bacterium in question has been heightened.

The grade of insusceptibility which pre-exists in any individual or species to any given infection we speak of as a natural immunity to that infection. Its production and perpetuation in the species are to be referred to the action of natural selection, and the survival of the fittest in the struggle for existence, just as are the other physiological and morphological distinctions which demarcate different species from each other. Like other race peculiarities, it varies in the extent to which it is developed in different individuals of the same species.

In contrast to the natural immunity of individuals or species—the refractory state, as it was called by Buchner—is the acquired immunity of convalescents from acute infection, which, being due to an individual reaction, is of necessity peculiar to the personality concerned. This form of immunity was naturally the earliest to attract attention, and, as I have already told you, attempts were made to procure immunity against small-pox, then the most terrible scourge of Western Europe, by imitating the method of Nature—that is to say, by variolation.

The means employed consisted in inoculating with material from the ripe pocks of persons ill of the disease, with the object of inducing a mild attack, and of thereby removing the chance, then almost a certainty, of subsequent more severe infection. But the results which followed were both variable and dangerous, for not only were the cases thus produced themselves infectious and capable of spreading the infection, but they were liable on occasion to develop the virulent disease from such inoculations, and death at times resulted. The procedure, therefore, was unsatistactory, and failed to gain a general acceptation.

It remained for Jenner to lay the foundation of all later progress in protective treatment by his most thorough and fruitful study of vaccinia, as the result of which, after more than twenty years of patient labour, he first employed in 1796 his method of vaccination, and two years later published his discoveries which made small-pox, then only less terrible in its devastation than the Black plague of the preceding centuries, a preventable disease.

The protection thus afforded was at first believed to be complete and permanent, but this has proved to be in part an error. The vaccination needs to be renewed at intervals in order to maintain complete protection. But it has been abundantly established that where inoculation is thoroughly performed and properly repeated at the necessary intervals—as, for example, in the Prussian army—variola has become unknown.

Little progress was made from the position reached by Jenner's labours until the latter half of the last century. The first advance was due to Nägeli and Buchner, who systematically investigated the question of immunity, and placed its phenomena in relation with the process of spontaneous recovery from acquired infections. They were speedily followed by Pasteur, who first prepared suitable vaccines for inoculation, and thus was able to procure in fowls immunity to fowl-cholera. Then, after a short delay, came the re-

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markable discovery of Behring, that the blood-serum of animals which have acquired immunity against a specific microbe is protective for other animals not otherwise immune.

The immunity thus induced by the injection of serum from immune animals was, however, found to differ from that which follows vaccination or recovery from disease in being only of a brief duration. It was named Passive immunity by Ehrlich, in contrast to the Active form produced by the reaction of the animal itself in an acquired infection.

Besides these forms of immunity, Ehrlich and others have further shown in certain cases the existence of inherited immunity in new-born offspring of those animals which have themselves acquired active immunity against a given disease. This inherited immunity is quite distinct from the hereditary immunity of a whole species to a particular infective agent, which, being naturally transmitted by the parents, is spoken of as a natural immunity. It is, as we shall see, transmitted by the mother only.

Accordingly we have to consider the following varieties of immunity:

Natural (hereditary) immunity.

Acquired immunity { Active: by recovery from inoculation or natural infection. Passive: by protective substances.

Inherited immunity from actively immune mother.

So far we have seen that in the process of recovery from an infective disease there is developed an immunity to that disease; further, that the immunity of any given animal to any given disease, whether it be natural or acquired, can be increased or diminished by events which modify favourably or unfavourably the general activity of metabolic processes. And it had been found in the course of the researches awakened by the opposition to the homœopathic school of Hahnemann and his disciples that one and the same agent may act now favourably and now unfavourably according to the amount or dose in which it is employed. These investigations led with other observations to a conclusion of extreme importance, clearly enumerated among the first by Hueppe, Arndt, and Schulz, that every agency which can in a certain concentration kill or injure the cell-protoplasm of a living body will in a smaller concentration, and on the other side of an indifferent point, act to the advantage of that protoplasm by rousing an increase of its activity.

This principle is fundamental both for experimental and for clinical medicine. It holds alike for the bacterial products and for chemical substances, and forms the basis of all useful treatment. It is this question of the concentration of the virus which determines whether infection shall provoke acquired immunity, or from the failure of immunity reaction end in death. We have already had a good example of these facts in the phenomena of localised bacterial inflammation where, in the focus of the inflammation, cell-necrosis is predominant, while at a distance, where the toxins have become diluted, cell-proliferation is the marked feature.

Following this principle of the concentration of the causal agent in disease, Pasteur in 1880 found that the bacterium of fowl cholera could be artificially reduced in its infective and intoxicating power, or, as we usually say, attenuated, so that on inoculation it produced not general infection and speedy death, but merely local and reactive inflammation. And animals thus treated were found to have become immune to the unattenuated micro-organism. These results were soon confirmed by numerous observers, who made use of a variety of methods of attenuating the bacteria which they employed. Among the methods used the most interesting was that of Duguid and Burdon-Sanderson, who discovered that the passage of a bacterium through the body of one animal often results in the attenuation of its virulence for a different species.

The immunity produced by such inoculations is specific, and in general affords protection only against the particular species of bacterium employed. And indeed it is not merely specific, but is also, to a great extent, peculiarly special against the particular variety of the bacterium concerned which has been used for the inoculation, as was shown by Herbert Durham of this hospital, and also by myself for the bacillus of enteric fever.

Though immunity was thus produced by the employment of attenuated cultures, yet it was thought that some degree of remaining virulence was essential, until Hueppe showed in 1887 that cultures which have entirely lost their pathogenetic properties can still induce immunity to the fully virulent bacterium. But it appeared most clearly

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from all the experiments that the infective agent used to induce the immunity reaction must always (leaving aside a few particular exceptions to which we shall return) be of the same character, that is to say, of the same species as that against which the immunity is desired. The immunity produced is therefore of specific, or more correctly isopathic, character.

After immunity has been induced by means of an attenuated micro-organism it can be further increased by subsequent inoculations with more and more virulent varieties of the same organism, until a condition of extremely high immunity is finally obtained.

The first and most successful application of these facts to medicine was made by Pasteur in 1885 in the case of rabies, with the result that the mortality among persons subjected to the infection from the bites of rabid dogs has been reduced in France from 16 per cent. to below one half per cent. It consists briefly in the consecutive inoculation of the patient with material from a series of spinal cords of increasing virulence until a condition of immunity has been produced. The spinal cords employed are those of rabbits killed by inoculation with a virus of a fixed and maximum activity, and are prepared for use by drying them for different periods and thus producing greater or less attenuation of the infective agent present in them.

Haffkine endeavoured to employ a similar method of protection by successive vaccination with two cholera cultures, the one attenuated, the other of exalted virulence, during the cholera epidemic in India in 1893 and 1894, though with only moderate success; and Wright, of Netley, has applied the method of vaccination against typhoid fever with excellent results.

After it had been shown by numerous researches that in the case of many bacteria pathogenetic action is due to the toxic action of soluble products which they liberate into the surrounding medium, it was discovered that immunity could also be produced by the injection of these substances apart from the bacteria, or by the injection of bacteria which had been killed.

There appeared, however, to be a striking difference between the immunity obtained by the injection of filtered cultured fluids or of dead bacteria and that produced by the inoculation of toxins artificially separated from bacterial cultures, in that immunisation with the former was protective only against the bacteria and not against their separated toxins, while the latter protected against toxins but did not confer immunity against the bacteria themselves. This discrepancy was due to the fact that, as we have already seen, the toxins as at first prepared were not in any sense the true bacterial toxins, but were artificial substances produced by chemical decompositions due to the action of the powerful chemical reagents used in their preparations. They were poisonous bodies, it is true, but they stood in no particular relation to the bacteria, whose products they were said to be. Accordingly immunity to these could not protect either against the bacteria or their natural products.

With the discovery of true bacterial toxins, such as the toxin of diphtheria as first prepared by Boer in 1896, and that of tetanus isolated by Brieger, Sidney Martin, and others, this difficulty disappeared, and it was proved that immunisation against the specific toxins of these organisms confers immunity not only against those toxins but against the bacteria themselves. The reason of this is easily to be understood. The bacteria in question produce their pathogenetic action through the agency of soluble toxins, which they secrete into the surrounding medium. If, then, an animal becomes immune to the toxins the bacteria have no further means of injuring it; that is to say, it is immune to the bacteria as well.

If, on the other hand, an animal be immunised against a bacterium freed from its soluble toxins, by filtration through porcelain, for example, it does not usually acquire immunity against those toxins, but only against the bacterium itself. This is because by the conditions of the experiment the animal is trained to rapidly destroy the bacteria introduced. Accordingly they are killed before they have had time to elaborate and secrete more than a very little toxin in the body, and the animal not being subjected to the action of much toxin, will obviously not be found immune to this if it be subsequently injected in large quantity. Thus animals immunised to tetanus spores were found by Vaillard to still remain susceptible to tetanus toxin, since the tetanus spores which the animals received contained no toxin, but only formed it after they had germinated into tetanus bacilli.

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We have, therefore the following results :—

Injection of	*Immunity to*
Soluble Toxins.	Toxins and Bacteria.
Bacteria.	Bacteria only.

What I have spoken of as the soluble or excreted toxins are usually called the extra-cellular toxins of bacteria. Besides these there are often present toxic substances in the bodies of bacteria—the intra-cellular toxins. To these is due the harmful action of the bacteria themselves, apart from excreted products; and immunisation against a bacterium means immunisation against these substances, which become liberated on the death and destruction of the micro-organisms. Since, however, in any culture of bacteria some are constantly dying and becoming broken up, a culture medium, even when freed from all bacterial bodies by filtration, will contain some, though it may be only little, of the intra-cellular materials as well as of the extra-cellular metabolic products.

Among the pathogenetic micro-organisms some exert their action chiefly, if not entirely, through the extra-cellular toxins, as, for instance, those of diphtheria and tetanus, while many others act in the main in virtue of their intra-cellular constituents, as in the case of cholera and typhoid among others.

Besides the methods which I have already dealt with, specific immunity may be induced by the injection of an immune-serum, which can confer passive immunity upon the animal injected. This great discovery was due to Behring and Kitasato, who showed, in 1890, that the blood-serum of animals actively immunised against tetanus or diphtheria has a qualitative or specific protective power for other animals, whether it be injected before the toxin, with it, or only at a later period. The classical research of Behring forms the basis of our modern serum-therapy, and has resulted in the saving of many thousand lives each year.

The power inherent in such sera of protecting against their corresponding toxins was described as anti-toxic, and is attributable to the action of a body or bodies present in the serum known as anti-toxin.

The immunity, which is produced by means of anti-toxin in contradistinction to that produced by vaccination with the bacterium itself, is of only comparatively brief duration. The protective substances injected, if not employed in the destruction of their corresponding toxin, are excreted in the urine, and otherwise disposed of by the animal body, and the immunity which has been conferred is quickly lost.

The immunity conferred by anti-toxic serum appears at once on the injection of the anti-toxin. It requires no lapse of time for its development; it must, therefore, of necessity be independent of any correlated action or reaction of the body-cells. Accordingly it was named by Ehrlich *passive* immunity, in contrast to the *active* form evoked by cellular reaction to the injection of bacteria or their products, and it is believed by him to consist merely in a transference to the animal injected of certain protective immune-substances, and is not in any sense whatever histogenous.

Up to this point we have spoken of all these sera as anti-toxic sera. It is, however, more convenient and preferable to confine the term anti-toxic sera to those sera which are especially active against toxins, applying the name anti-microbic sera to such as have their action on the bacteria themselves, and using the general term of immune sera to include the two.

Immune Sera.—Anti-toxic sera, containing anti-toxin for the bacterial toxin. Anti-microbic sera, assisting the destruction of the bacteria themselves.

The methods of producing an immunity hitherto discussed are of specific nature, and result in a specific protection. There are, however, also certain non-specific means by which immunity can also be induced. So long ago as 1877 Pasteur had noted that if animals susceptible to anthrax be inoculated with a mixture of this bacillus and other bacteria, the anthrax is unable to produce disease, and many similar discoveries soon followed. And it was shown, for example, that inoculations with the bacillus pyocyaneus can evoke immunity to anthrax. These observations led to the enunciation of two different views in explanation. The first was due to Hueppe, who held that the result was due to stimulation and increased activity of the normal protective processes or substances of tissue cells. The second, that of Klein, was that in cases of this kind the bacterial toxins were identical, and consequently inoculation with one variety of bacterial products resulted in protection also against the other. To this question I shall presently return.

It was, moreover, shown by various observers

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that in many cases cultures of pathogenetic bacteria could be destroyed by the agency of saprophytic forms. Hence arose an endeavour to seek a cure for the bacterial diseases in a therapeutic use of saprophytes. Cantani was the first to venture on this method of bacterio-therapy, using bacteria of putrefaction in an attempt to induce the healing of tuberculosis. He was followed by many others, among whom I need only mention Coley, who introduced the injection of the *products* of streptococci in a solution known as Coley's fluid for the treatment of inoperable malignant growths. Here, also, any result that is attained must be attributed to a non-specific stimulation of the normal protective processes. In what such stimulation may consist can, to some extent, be gathered from a consideration of the results of inoculation with heterogeneous, that is, non-bacterial substances.

Already, in 1888, Dr. Wooldridge of this hospital had found that he could by the injection of tissue fibrinogens obtain protection against anthrax. Then it was found that similar protection followed inoculation with digestive ferments in the case of cholera, with spermin, with emulsin, with cinnamic acid, and numerous other bodies, and finally with such simple substances as broth or normal salt solution.

Now Issaeff has shown that the uniform result which follows these injections is the production of remarkable leucocytosis, and that the appearance and duration of the immunity coincides with occurrence and persistence of the leucocytosis. And Bordet has found by other methods that at the same time phagocytosis of bacteria is much increased coincidently with the increase of leucocytes.

All the methods, then, which give rise to non-specific protection have this in common—that they produce a marked leucocytosis. So far as information goes they have no other common factor. There is, therefore, evidently some close relation between the two events leucocytosis and bacteriolysis.

This observation is of great importance for the phagocytic theory of immunity, of which I shall speak directly. With it may possibly be correlated the clinical success attending laparotomy for early peritoneal tuberculosis, and the production of a local congestion by Biers' method in tuberculosis of extremities. It probably also bears upon the doctrine of Rokitansky that lung tuberculosis is rare in conditions associated with lung-congestion, but common in those leading to chronic anæmia of that viscus.

So far we have seen that artificial acquired immunity can be induced by the following methods:

Acquired immunity—

- Specific
  - bacterial inoculation.
  - inoculation of bacterial products.
  - inoculation with specific immune serum.
- Non-specific
  - inoculation with other bacteria or their products.
  - with heterogeneous material.

The specific acquisition of immunity by natural disease is evidently the same process that occurs after inoculations in the laboratory, and leads to the appearance of the same protective substances in the serum of the convalescent. In the same category comes the inherited immunity of which I have already spoken. This is transmitted only through the mother, and is acquired in utero by the fœtus. *Passive* immunity can also be transmitted to the offspring by the mother's milk if she be rendered actively immune.

We must now proceed to a consideration of some of the more important theories which have been advanced in explanation of the phenomena of immunity. Of these the earlier suggestions only need a cursory reference.

The view of Pasteur commonly spoken of as the *Exhaustion* theory was that bacteria in their growth within the body require the use of, and eventually use up or exhaust, some pre-existing substance of the body which is necessary to their propagation. This substance having been exhausted, the body ceases to afford a suitable culture medium for their cultivation, and is in consequence immune.

With the discovery of Behring that immunity can be transferred from an immune animal to the unimmune by transference of serum, this theory became at once untenable.

Similarly the *Retention* theory of Chauveau according to which the immunity following inoculation or disease depended on the retention in the body of some hypothetical bacterial product inimical to the growth of the bacterium in question and thus protective from a fresh invasion, although in harmony with the observation that the excreta of

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bacteria, as of other animals, are in general deleterious to their well-being, was yet entirely unable to explain how an immunised animal after bleedings amounting in a brief period to more than the total normal blood-content of that animal could still continue to possess and yield an immune serum of practically undiminished power; though this was shown to be the case by Ehrlich.

More fortunate was the *Adaptation* theory of Grawitz, which attributed the phenomena in question to a gradual adaptation of the body to resist the injurious action of the invading micro-organisms. This view of Grawitz may be looked on as in a measure the parent of the modern cellular theory of immunity.

Coming, then, to the more recent views, we are concerned with three chief features of immunity:

1. The Cellular or Phagocytic theory of Metschnikoff.

2. The Humoral theory of Grohmann (1884) and his supporters, Flugge, etc.

3. The Humoro-cellular theory of Bouchard.

These we shall now consider in their order and mutual relation.

Cellular theory.—So long ago as 1864 Traube and Geschleiden showed that the animal body quickly disposes of bacteria introduced into it by artificial means. Such a power must depend in essence more or less directly on the protoplasmic functions and activities of the body cells.

Again, the local reaction to local bacterial infection, just as to other causes which lead to local injury of tissue without involving its immediate destruction and death, was seen to consist in the process of inflammation which we have already discussed. Now in this process, as we have seen, the wandering cells of the body collecting at the focus of infection are found to be engaged in actively attacking the invading micro-organisms. Certain, also, of the fixed cells of the tissues possess this so-called phagocytic power, and play their part in the reaction concerned. Accordingly Metschnikoff was led in 1883 to the conclusion that on the functions of the phagocytes depend the essential elements of protection. To this class of phagocytes belong the following varieties of cells, as Metschnikoff believes: Macrophage (large mononuclear) and microphage (polymorphonuclear) leucocytes of the blood, wandering cells of the tissues, connective-tissue corpuscles, endothelial cells, cells of the spleen pulp, and the sarcoplasm of voluntary muscle fibres. This group of the phagocytes can be traced downwards through the animal series to the earliest forms in which differentiation of function first appears among the cells of the organism, and still lower the same properties are exhibited by cells of yet undifferentiated function, and finally by the simple unicellular protozoa themselves. We have, then, here a cell-reaction invariably present and universal throughout the animal kingdom from protozoa up to man, and which might well serve as the natural basis of immunity. The production of immunity would then consist, as Metschnikoff believes, in the gradual heightening and extension of this natural reaction.

Next followed the discovery of Stahl and Pfeiffer, made originally on unicellular plants in 1884, of the phenomenon of chemotaxis, and it was found that the determination of phagocytes towards the centre of infection was the result of chemotactic influence or allurement exercised on them by the soluble products of the bacteria in question.

It was not clear, however, whether the bacteria taken up by phagocytes were killed by them, or whether they had first been killed by other agencies and only subsequently ingested by the phagocytic cells like any other inert foreign substance. And it was held, in opposition to Metschnikoff, that only dead bacteria were thus attacked by leucocytes. Lubarsch then proved that living bacteria are actually taken up, and, indeed, in immune animals are taken up more quickly and in greater numbers than are dead ones.

But it was now seen, on the other hand, that the bacteria thus ingested by the white cells are not invariably nor necessarily killed by them. Thus Trapeznikoff showed that the spores of anthrax may live on within the leucocyte until the latter dies or is killed, and then proceed to develop and produce their usual effects; and similar facts are true for tubercle bacilli and the giant-cells of tubercles.

Further, Grohmann had shown in 1884 that cell-free blood possesses the power of destroying bacteria introduced into it. This was confirmed for anthrax by Fodor in 1887, and Nuttall in the following year published a whole series of experiments on the defibrinated blood of certain animals which he showed possessed a bactericidal power for anthrax.

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A TEXT-BOOK OF MEDICINE. By C. H. FAGGE, M.D., and P. H. PYE-SMITH, M.D., F.R.S. 4th edition, 2 vols. J. & A. Churchill, London, 1902.

IT is well known that when Dr. C. H. Fagge died at the age of 45 in November, 1883, he left behind him the almost completed manuscript of a text-book of medicine. The medical profession owes to Dr. Pye-Smith an enormous debt of gratitude, for he not only added the section on diseases of the skin, and arranged for the filling-up of some lesser gaps, but he edited and saw through the press the whole work, which was published in 1886. At once those who read it prophesied that a book which would prove to be a classic had appeared, and this prophecy has been verified. The recent text-books of the time were a dry record of facts, but Fagge wrote descriptions which the reader read for the mere pleasure of reading. He showed a strikingly wide knowledge of the literature of medicine, and what was better still, he possessed a judicial mind which enabled him to sort wheat from chaff. He checked his own and other authors' statements by constant reference to the records of Guy's Hospital and to his own clinical and post-mortem experience, and with rare ability he criticised the theories then prevalent. The reader when he laid down the book could not help saying what a wonderful achievement for a busy man æt. 45. But Dr. Pye-Smith has done more for us than enable us to read this first edition. He has taken charge of the book since 1883, and has brought out three editions subsequent to the first. The fourth has just been issued, and it is impossible in the space at our disposal to review in detail the whole of the two large volumes. But we can say they are charming reading. In consequence of the great advances in medicine during the last twenty years Dr. Pye-Smith has been compelled to omit parts of Fagge's original writing in order to find room for the necessary additions; in other places the original is considerably modified. We quite agree with Dr. Pye-Smith that the history of any disease is of great use in enabling us to know about it thoroughly, and it is in the historical part that this book is particularly strong, and the many classical quotations whet the reader's appetite. In a book this size it would be easy for the reviewer to point to what he would have otherwise. For our part, as examples we should have liked a fuller discussion of the various theories that have lately been put forward about diabetes, or, again, a rather more important account of pancreatic cysts and acute and chronic pancreatitis. In this edition, as in the first, the account of nervous diseases strikes us as the least adequate. In the first edition it read as though it had been written many years before Dr. Fagge's death, so that by now the plan of it may be thirty years old, and in our opinion it is hardly the mode of arrangement most suited to modern neurology.

On the other hand the sections on diseases of the skin, intestinal obstruction, typhoid fever, and Bright's disease are admirable. But whatever sins there may be in this fourth edition, they are sins of omission rather than commission, and we can confidently recommend this book to our readers as one which will be read with pleasure and sustained interest, and which gives a scholarly account of the science and art of medicine in good English. It is well and clearly printed, but we do wish the publisher had followed the example of other publishers, *e. g.* Messrs. Macmillan, and had put the date of the publication of each of the other editions. As far as we know Messrs. Churchill do not do this with any of their books, but in our opinion it is very desirable, as it adds much to their interest.

WE have received from Burroughs, Wellcome, and Co. a specimen of the "Wellcome" brand of pilocarpine nitrate. The considerable variation in physiological action exhibited by the galenical preparations of jaborandi has led to experiments which have proved that the full physiological effect of jaborandi is produced by pilocarpine, and, further, that the nitrate is a suitable salt of the alkaloid for medicinal purposes. Pilocarpine nitrate "Wellcome" brand is issued in tubes containing gr. 15 and gr. 60, in bottles of ½ oz. and 1 oz. respectively.

We have also received a specimen bottle of "Tabloid" kino compound powder, B. P. gr. 5 (0·324 gm.). Kino is well known to be an astringent of considerable service in the treatment of certain forms of diarrhœa and dysentery. "Tabloid" kino compound powder will be found to retain in a marked degree all the properties of freshly-prepared compound kino powder, and to be a convenient preparation to employ. Each product represents five grains of the official powder.

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THE EFFECT OF PREGNANCY ON CHRONIC HEART DISEASE.*

By CUTHBERT C. GIBBES, M.D., M.R.C.P.,

Physician and Pathologist to the Hospital for Diseases of the Heart, London.

GENTLEMEN,—Having done me the honour of electing me your President for the ensuing year, my first duty, after tendering you my sincere thanks, is to deliver an address. The choice of a subject suitable for this occasion has been attended with some difficulty, for however interesting certain lines may be to oneself, they have not the same interest to others. Moreover, this Society being a clinical one, it behoves me to limit myself as much as possible to practical detail, and only touch lightly on theoretical ones.

Having had many opportunities of studying the effect of pregnancy on chronic heart disease, it occurred to me that this subject would have a more general interest than any other coming within the range of my own special work. The majority of the articles written on the subject have been contributed by obstetricians, and therefore heart disease has been looked upon as a complication of pregnancy. I propose to take the opposite line, and treat it from a cardiac point of view. It is a comprehensive subject, and contains many disputed points. I do not, however, intend taking up your time by entering into any discussions, and shall therefore adopt the views that are now generally held.

First, I shall consider certain physiological and pathological conditions which may arise during a healthy pregnancy; secondly, the complications and prognosis of chronic heart disease apart from pregnancy; and thirdly, the conduct of the diseased

* Presidential Address to the Chelsea Clinical Society on October 28th, 1902.

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heart when pregnancy is present, and the treatment best adapted to such circumstances.

There are certain normal changes that take place in the cardio-vascular system as a result of pregnancy. These may be summarised as follows:

1st. The vascular area is increased in consequence of the development of uterine blood-vessels and the dilatation of those in the surrounding tissues.

2nd. The quantity of the blood is increased during the last four or five months of pregnancy.

3rd. The quality of the blood is altered.

a. The relative amount of water, the white blood-corpuscles, the fibrin, and the extractive matters are increased, especially during the later months of pregnancy.

b. The red blood-corpuscles, the hæmoglobin, and the albumen are diminished, but rapidly increase after parturition.

4th. The arterial tension is increased during pregnancy and puerperium, but the greatest tension is during labour.

5th. The systemic and pulmonary veins become engorged, especially those of the lower extremities.

6th. The heart gradually becomes enlarged, and increases in weight (about $\frac{1}{6}$) owing to dilatation and hypertrophy. The left ventricle is more hypertrophied than the right, especially during the later months of pregnancy. The right ventricle, on the other hand, is dilated to a proportionally greater extent than the left, the conus arteriosus, according to Stengel and Stanton, frequently presenting marked evidence of dilatation. The apex-beat becomes gradually more displaced towards the left, and during the later weeks of pregnancy has a pushing character. The heart's action is not increased in frequency during pregnancy. Systolic murmurs may be heard over the pulmonary and tricuspid areas, but there is no evidence to prove that the cardiac dilatation produces mitral insufficiency under normal conditions, the murmurs described by Angel Money being probably of tricuspid or pulmonary origin.

The first stage of labour has little, if any, influence on the normal heart beyond quickening its action, but during the second stage the strain is severe, and the tension in the venous system is increased by the addition of the blood expelled from the uterus during its contraction. Each bearing-down pain is necessarily accompanied by a violent expiratory effort, with closure of the glottis, producing a positive pressure on the heart. This pressure prevents the increased venous tension dilating the right auricle, and may even be sufficient to render the pulse imperceptible for a few beats. The thin right auricle being especially compressed and the return of blood to the heart delayed, the tension of the whole venous system becomes still further increased. The expiratory effort is followed by a prolonged inspiration which renders the intra-thoracic pressure sub-atmospheric, and thereby exerts a suction influence on the distended veins. The result is that the right auricle and ventricle are speedily over-distended with blood, to be relieved immediately afterwards by the next expiratory effort. When a pain ceases the uterus relaxes, and lessens the effect on the heart by abstracting a large quantity of blood from the vascular system.

After delivery the apex of the heart soon returns to its normal position. The left ventricle becomes smaller, but the right side of the heart only gradually resumes its normal size. Some authorities state that there is a rapid diminution in the arterial tension after delivery. No doubt the stress of parturition and loss of blood, even within normal limits, may lower the tension, but I do not believe that any physiological lowering of the arterial tension occurs immediately after a purely normal labour. Systolic murmurs are frequently heard after delivery as well as before, but they probably all have the same origin.

7th. There is little, if any, diminution in the vital capacity of the lungs, the displacement upwards of the diaphragm by the enlarged uterus being neutralised by the widening of the base of the thorax. The tension in the pulmonary blood-vessels is much increased.

8th. The kidneys become enlarged and the urine altered in composition. With the exception of chlorides, the solids are all diminished in quantity, and a trace of albumen is present during the later weeks of pregnancy in about 2 per cent. of all cases. Many class this as a physiological albuminuria, and I therefore now refer to it as such, but I think it is far safer to regard all pregnancy albuminurias as pathological.

9th. The thyroid gland, liver, and spleen become somewhat enlarged and heavier.

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Although I have enumerated the above views somewhat dogmatically, I do not for one moment wish you to think that they represent an unanimous verdict of opinion, because such is not the case. Differences of opinion exist on many points, but I believe my summary represents the verdict of the majority.

Having thus laid down what may be termed a normal standard, I shall now refer to certain pathological conditions that may present themselves during the pregnancy of a previously healthy woman.

When all the organs of the body are in a normal condition, the strain of pregnancy falls on the kidneys, and to their failure may be attributed the chief complications that arise in the healthy subject. Herman divides the renal diseases arising from pregnancy into three groups. 1st. A passive congestion of the kidney, arising from the pressure of the gravid uterus, and not productive of eclampsia. In my opinion the trace of albumen that is frequently found during the later weeks of pregnancy comes under this heading, but it has no clinical significance. In rarer instances, albumen may be present in an appreciable quantity, yet no casts or other evidence of structural disease of the kidney exist. 2nd. A chronic renal disease peculiar to pregnancy. Saft defines this as a disease of the kidney which takes rise in pregnancy alone, never leads to important disturbance of the general health, and in childbed quickly subsides. 3rd. An acute renal disease peculiar to pregnancy. This disease is not a nephritis, but an acute degeneration of the renal epithelium, and is the cause of eclampsia.

In these three groups the albuminous precipitate chiefly consists of paraglobulin, serum albumen being present only in small quantities. When renal disease has existed previously to and independently of pregnancy, serum albumen predominates or is present alone, the occurrence of serum albumen in the earlier months, or its presence at any time in larger quantities than paraglobulin, always suggesting that the renal disease existed before the patient became pregnant. The distinction between the two kinds of albumen is made by saturating a portion of urine with magnesium sulphate. This precipitates the paraglobulin, but not the serum albumen; the urine is then filtered and the filtrate tested for the latter in the usual manner.

Various causes have been assigned for the occurrence of the renal diseases of pregnancy, and the subject has been fully discussed elsewhere; but no unanimity of opinion exists. It seems probable, however, that they are the result of an association of events rather than one individual cause. Herman considers it more than possible that the acute condition is due to the introduction of a poison from without.

The two points that stand prominently forward as predisposing, if not exciting causes, are—1st. The pressure of the gravid uterus on the renal veins impeding the venous return from the kidneys. 2nd. The accumulation of toxic materials in the maternal blood-vessels, owing to the defective elimination of fœtal waste products.

I have entered into this portion of the question because I consider these renal diseases have a direct influence on the heart. Eclampsia is, of course, in itself a complication to be greatly dreaded; but it is not the only one, for a toxic blood-supply is liable to cause degeneration, even in healthy heart-muscle, producing dilatation of the heart and failure of compensation; consequently its effects on an already diseased heart must always be seriously considered.

Turning now to the heart affections that may arise *de novo* during pregnancy, we find that some authorities believe that pregnancy may of itself cause acute and chronic endocarditis, but the weight of evidence tends to prove that it never does. Malignant endocarditis may, of course, arise from some septic condition of the generative organs, and acute rheumatism, chorea, scarlet fever, etc., may give rise to an inflammatory lesion of the heart in those who are pregnant equally with the non-pregnant, but the former do not appear to be specially susceptible, nor are they more liable to recurrent attacks of endocarditis than others.

Handfield-Jones considers that an apparently normal heart may become dilated as a result of pregnancy, and failure of compensation supervene (I use the term "apparently normal" because it is often difficult, if not impossible, to be certain that the heart-muscle is in a normal condition). The increased quantity of blood in pregnancy necessitates an increase in the size of the chambers of the heart; the dilatation thus produced is met by a corresponding amount of hypertrophy (chiefly of the left ventricle), the balance is struck, and a

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perfectly compensated heart results. Should, however, the left ventricle fail to hypertrophy, cardiac insufficiency results, and dilatation manifests itself by shortness of breath, palpitation and œdema of the lower extremities, premature expulsion of the contents of the uterus taking place about the third or fourth month, or later if the dilatation is less pronounced. Cases of this nature are seen occasionally, but I very much doubt if they are met with so frequently as he would lead us to suppose. In a patient who miscarried at four and a half months, I had carefully examined the heart eight months before the miscarriage occurred, and, as far as clinical diagnosis can be relied on, it was perfectly normal. It became much dilated during pregnancy, and returned to its previous condition after abortion took place. If these cases only occurred in young anæmic women with flabby hearts it would be easily understood, but it is not so, for all the cases I have seen have been in older women, their ages ranging from twenty-five to forty. I quite agree that the immediate cause is the absence or insufficiency of the normal hypertrophy of pregnancy, but I believe that in many cases there is some pre-existing change in the heart-muscle that prevents it from responding to the fresh demands made upon it.

Handfield-Jones also assumes that a somewhat similar process takes place in the normally hypertrophied heart after parturition as that known to occur in the uterus. He believes that "in the majority of cases the hypertrophied muscular fibres of the left ventricle do, soon after delivery (and in some cases even earlier), undergo a process of retrograde metamorphosis, and that the change is fatty in nature."

There is no doubt that the normal heart-muscle is occasionally found during the later weeks of pregnancy in a dangerously weakened condition; whether a fatty granular degeneration is present we may have no evidence to prove, as fatal results are warded off by timely interference. We also know that fatty degeneration has been frequently found on post-mortem examination. But I do not think it necessarily follows that these conditions are the result of a premature cardiac involution, as Handfield-Jones suggests. Degeneration of heart-muscle, as a rule, has a gradual onset, but we find that diphtheria is specially marked by the rapidity with which the heart-muscle may become involved. It appears to me, therefore, to be more probable that the toxins of pregnancy have a similar effect.

Having thus considered certain complications that may occur as the result of pregnancy, I shall now discuss the complications that may be looked for in heart disease not the result of pregnancy. I shall not take up your time by speaking of congenital disease or lesions confined to the right side of the heart, because it is extremely rare to meet with pregnancy under these conditions, and I have no personal experience on the subject. I will therefore confine my remarks on valvular disease to affections on the left side of the heart, referring more especially to the mechanism of those diseases and the result of failing compensation.

In pure mitral stenosis we have slowly progressing constriction of the mitral orifice, which renders the process of filling the left ventricle more and more difficult as the disease advances. In the early stages the left auricle becomes hypertrophied, and is able to carry on the work, but as the disease advances and the mitral orifice decreases in size, the tension behind the constriction proportionately increases, and the left auricle fails. The right ventricle and pulmonary arteries become dilated and hypertrophied, and gradually take on the work, aided to a great extent, in some cases, by the diastolic suction power of the left ventricle. On the right ventricle, however, devolves by far the greater portion of the work, and the entire responsibility when failure occurs. This being a progressive disease, the tension in the right side of the heart gradually increases, the venous circulation becomes more and more involved, the various organs of the body passively congested, and their fibrous tissue increased.

In mitral insufficiency the venous circulation suffers in a somewhat similar manner, the complications being in direct proportion to the amount of blood regurgitated. The mechanism of the disease consequently differs from that of mitral stenosis. The left ventricle is dilated and hypertrophied, whereas in stenosis it is seldom, if ever, dilated, although it may be hypertrophied. On the other hand, the left auricle becomes dilated by the reflux of blood, but is not hypertrophied to the same extent as in stenosis, in consequence of the freedom with which it is able to get rid of its contents. For the same reason the left auricle,

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the pulmonary vessels, and the right ventricle are subject to a less intense and continuous strain, and are therefore less liable to degenerative processes than in stenosis.

When mitral obstruction and insufficiency are combined the mechanical results are the same as in stenosis. It must not, however, be supposed that strict clinical lines of demarcation can always be laid down between these three conditions. Pure mitral stenosis is by no means common, for the absence of the systolic portion of a double mitral murmur does not necessarily imply the absence of regurgitation, nor does the presence of systolic murmur only necessitate the absence of stenosis, for the presystolic portion of the combined murmur is frequently absent when the heart is badly failing.

Mitral regurgitation is a common result of valvular insufficiency from dilatation of the mitral orifice; but, as a result of inflammation of the mitral cusps, pure regurgitation is rare, for there is generally some stenosis present, and a process of contraction of the cusps is liable to take place, the case ultimately assuming the clinical aspects of obstruction.

I have been much struck when reading recorded cases of pregnancy in heart disease to find what a large proportion of patients have had mitral insufficiency, this diagnosis having been made because a systolic murmur was alone heard at the apex. Consequently mitral insufficiency is looked upon by some authorities as a greater source of danger to pregnant women than aortic disease. This large proportion of adults suffering from failing compensation in mitral regurgitation (we are not dealing with anæmic girls) is contrary to my experience in the out-patient department, and is not borne out by post-mortem statistics.

I do not believe insufficiency is solely present in these cases, but that mistakes have been made through attempting to diagnose the heart condition from one physical sign alone.

Although the mechanism of these mitral diseases is different, their influence on the general system is the same, the difference being only one of degree.

The complications that more especially concern our present subject are those affecting the heart-muscle, the lungs, and the kidneys.

It is of the greatest importance in the prognosis of heart disease to determine the condition of the ventricular muscle; unfortunately, however, this is by no means an easy thing to do. In mitral disease it is especially difficult, for you may have two cases presenting almost exactly the same physical signs, and having the same surroundings, yet one readily yields to treatment and the other does not, the different results being due to the condition of the heart-muscle. It is the same when women with similar diseases of the heart become pregnant; one may die from her first or second pregnancy, while another has six or eight pregnancies, and yet the heart recovers itself to a great extent, the condition of the heart-muscle being the only primary difference between them. This failure of heart-muscle may be due to fibrosis or fatty degeneration.

Cardiac fibrosis may have arisen from the original endocarditis or from any recurrent attack, or may be superadded to any valvular disease, in consequence of prolonged congestion of the coronary veins, or as a part of a general arterial sclerosis.

Fatty degeneration always arises from defective nutrition, and as a complication of valvular disease it is only present during the later stages. In aortic disease it results more particularly from insufficiency in the quantity of blood supplied to the muscle itself, from narrowing of the coronary arteries; whereas in mitral disease it is more especially caused by a deterioration in the quality of the blood, in consequence of pulmonary and renal complications. Fibrosis and fatty degeneration may, of course, occur independently of valvular disease, the prognosis being then equally grave.

Thrombosis is a source of danger in both pregnant and non-pregnant women—cardiac thrombosis having a more especial reference to our present subject. Cardiac thrombi are formed when compensation is failing, their most frequent sites being the auricular appendices and the apices of the ventricles. They may prove a source of danger when retained in the heart itself, by reason of their size or position, but the most frequent danger arises from their becoming detached and forming emboli. In the case of the lungs these emboli come from the right side of the heart and the pulmonary artery, and the commonest site is the lower lobe of the right lung. In the arterial system they come from the pulmonary veins or the

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left side of the heart, the kidneys, spleen, and brain being the most frequent sites. The main trunk or primary branches of the pulmonary artery may be the site of thrombosis or embolism; the symptoms in both cases are identical, and sudden death an equally common result.

Passive congestion of the lungs is a constant result of mitral disease, and predisposes them to attacks of broncho-pneumonia. As the tension increases in the pulmonary blood-vessels, their walls become dilated, thickened, and atheromatous, and hæmorrhages are likely to occur. The fibrous tissue of the lungs increases, and a condition of brown induration may be present. When compensation fails the bases of the lungs become œdematous, and bronchial catarrh is almost continuously present from backward pressure in the bronchial veins. The blood is consequently insufficiently oxygenated and the general nutrition suffers. The lungs are much more seriously implicated in stenosis than in insufficiency, hæmorrhages, infarctions, and emboli being oftener met with in the former than in the latter disease. The kidneys do not, as a rule, become involved until late in the course of the disease, after the lungs have become affected, and when compensation has failed. In the early stages it is simply a passive congestion, but when the conditions have persisted for a longer period of time there is an increase in the fibrous tissue, and a condition of what has been termed cyanotic induration is present.

In aortic incompetence the left ventricle is both dilated and hypertrophied. As long as the blood-supply to the muscle is sufficient for its nourishment the work is carried out satisfactorily, but when the hypertrophied muscle outgrows its blood-supply, fatty degeneration sets in and compensation fails; if life is prolonged the ventricle dilates and the mitral valves become insufficient. There may be some increase in tension of the pulmonary veins on account of the left auricle failing to empty itself sufficiently, but lung symptoms do not become a serious item in these cases unless the mitral valves are insufficient. The kidneys are rarely involved as long as the mitral valves are effective; should, however, the mitral orifice dilate and the valves become insufficient, cyanotic induration may be present in the same manner as if the mitral was the primary disease. In aortic insufficiency the aorta and the arteries generally are subject to intermittent high tension at each cardiac systole; this ultimately produces degeneration of their coats, but the complications resulting from these causes have no special bearing on pregnancy unless the degeneration is far advanced.

In compensated aortic stenosis the hypertrophy of the left ventricle bears an inverse proportion to the constriction of the aortic orifice, and dilatation only results from degeneration of the muscular walls. When the orifice is much constricted epileptiform attacks and syncope may occur and cause sudden death, but if mitral insufficiency is present the symptoms are moulded on that type of disease.

Death in mitral disease usually results from asthenia, the general venous congestion making itself felt in the various organs of the body, sometimes one and sometimes another bearing a preponderating influence in hastening the end. Their combined influences produce a gradual deterioration in the general nutrition, the heart-muscle, on which the whole question turns, thereby becoming degenerated. Sudden death is the exception, but it may occur from thrombosis or embolism of the pulmonary artery. As this probably never takes place unless degeneration is present, death may be said to be solely due to myocardial degeneration.

In aortic insufficiency, on the other hand, sudden death from asystole is not an unfrequent occurrence in pure and complicated cases, but it rarely occurs without some premonitory signs of heart failure, although they may not have been recognised by the patient. If the mitral orifice has been involved by the original attack of endocarditis, or insufficiency results from secondary dilatation, sudden death is not common, the train of events being moulded by the mitral element, and death ensuing from asthenia, as already described.

The prognosis of chronic valvular disease depends mainly on two points—the position in life of the patient, and the condition of the heart-muscle; the nature of the valvular lesion being a minor consideration. We find, however, that the heart-muscle is more liable to fail in some forms of valvular disease than others, the relative duration of life varying in consequence. Commencing with the least dangerous, in my opinion, they bear the following relations:—Mitral insufficiency, aortic obstruction, mitral stenosis, aortic insufficiency, and aortic and mitral disease combined.

(*To be continued.*)

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THE DIAGNOSIS AND TREATMENT OF INFECTIVE THROAT DISEASE.

An Address delivered before the Wimbledon Medical Society.

By F. FOORD CAIGER, M.D., F.R.C.P.

(*Concluded from p.* 168.)

Now I will add a few words on the treatment of scarlet fever. Many are the drugs that have been tried with the object of exerting a controlling influence over the course of the disease, but unfortunately it must be confessed that even at the beginning of the twentieth century we do not know of any single drug which is capable of exerting any specific effect. Numerous are the drugs which have been tried, from old-fashioned belladonna down to relatively modern products, such as thymol, salol, and even izal; but as a matter of fact there is no evidence that any of these drugs are capable of exerting any controlling influence on the progress of the disease. It was thought at one time that we had a specific remedy in antistreptococcic serum. Baginski, of Berlin, claimed to have reduced the mortality of his septic scarlet fever cases to 14 per cent. Considering our normal mortality in this class of case, his claims for antistreptococcic serum will not, I am afraid, recommend themselves very strongly to us. I can only say with regard to antistreptococcic serum that I have tried it thoroughly, and there has not been the slightest benefit apparent from its use. Some years ago, at the instigation of the late Dr. Bristowe and Dr. Carne Ross, I treated a series of cases with a decoction of cinnamon. The latter gentleman thought that this drug had a controlling influence on the progress of scarlet fever in cases which could be brought under treatment at a sufficiently early date. It was therefore tried religiously in a series of two hundred consecutive cases, with the result that the mortality was the same as that of the previous two hundred, but the incidence of complications in the cinnamon series was very much less than is usually the case. To this extent, then, credit must be given to cinnamon, namely, that its exhibition was attended with a distinct diminution in the number of the ordinary complications of the disease.

As regards the throat, it is extremely important that efficient local treatment should be adopted, especially in cases of the septic variety. In many ordinary attacks, however, there is a tendency for the tonsils to become ulcerated, and by means of efficient local irrigation you may be successful in preventing the case from becoming septic. I would strongly emphasise the importance of antiseptic irrigation of the fauces in scarlet fever except in the very mildest cases. In certain of the more severe septic attacks the ulceration is not confined to the tonsils, but involves also the pillars of the faucial arch and the folds of mucous membrane joining the base of the tongue and the epiglottis and upper lateral borders of the larynx. As a result of this, the proper movements of the palate and epiglottis are interfered with, and deglutition becomes both difficult and painful, each act of swallowing being attended with the passage of food and offensive secretions through the laryngeal orifice. In this way the patient is liable to frequent fits of choking, and, moreover, the supervention of septic bronchitis or broncho-pneumonia is to be feared. Under these circumstances the food should be administered by means of a nasal tube, or the patient for a time may be entirely fed by rectum. Occasionally the amount of swelling of the epiglottis and adjacent tissues is so great as to impede respiration, and the breathing becomes stridulous and painful. Should the nares at the same time be blocked, as is so often the case, the patient is unable to obtain any rest whatever, since, the moment he falls asleep, the epiglottis and base of the tongue tend to fall back and obstruct the upper aperture of the larynx. The avoidance of this necessitates a conscious act of adjustment on the part of the patient, and consequently, the moment he lapses into sleep, he is waked up again half choked and suffocated. For the relief of this distressing condition tracheotomy is imperatively demanded. The adoption of nasal feeding, *plus* tracheotomy, is often attended with the best results, since the patient will thereby be enabled to obtain nourishment and sleep, both of which are so essential to his recovery. The tissues, moreover, are kept at rest, whereby their repair is undoubtedly facilitated.

As regards external treatment of the throat in scarlet fever, in severe cases I think everybody will admit that there is a great deal of comfort to

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be derived from hot applications, but whether fomentations or linseed meal poultices are employed does not seem to matter very much. Perhaps a poultice is more effective than a fomentation in promoting comfort, and it is probably not without influence on the resolution of the tender and inflamed glands which contribute so much to the patient's distress. Whether its influence be in the direction of resolution, or whether it act by hastening the process of suppuration is immaterial, as the consequent result to the patient is in either case a beneficial one. The continued application of a hot fomentation or a linseed meal poultice certainly tends to diminish the pain and swelling. It should be changed frequently, and, of course, properly made and evenly applied. Local discomfort may often be lessened by frequently sucking small pieces of ice, but in some persons more relief is obtained when the fluid nourishment is taken as hot as it can be borne.

In respect to the restlessness which is so often present, especially with a high temperature, there is nothing so beneficial, so far as I know, as efficient sponging of the surface. I would not advise that the sponging should be done with quite cold water in the depth of winter, but in summer-time cold water may be used with advantage. I do not advocate sponging so much with the idea of lowering the temperature as of promoting quiet sleep. It is advisable to tepid-sponge the patient every evening as a matter of routine, for by so doing one not only lessens the tension of the skin, but the restlessness is materially reduced. A wet pack in the same way acts as an excellent sedative, but I do not often recommend either sponging or the wet pack for its direct antipyretic effect.

The diet during the febrile stage should mainly consist of milk and beaten-up eggs, but the addition of a little soup, beef tea, or mutton broth forms a pleasant variety, and will do no harm. There has long been a tradition that too much care cannot be insisted on with regard to the diet in the early convalescence of scarlet fever; but we find practically that as soon as the temperature has fallen to the normal, and the patient is ready to swallow it, a more solid diet is permissible. In scarlet fever the fact of the patient being ready to swallow it may be taken as the most reliable indication of his fitness for solid food. Bread soaked in milk, bread and butter, milk puddings, and custards may then be added; next a little fish, then, in a day or two, meat; so that within a week from the time of the temperature falling to normal the patient may be allowed to take his ordinary food.

Now in regard to the treatment of the patient during early convalescence, it is very desirable that warm baths should be given frequently, not only in order to facilitate the process of desquamation, but also in order to diminish as far as possible the risk of renal complications; for in proportion as the skin is encouraged to act, the renal tension will fall, and the patient be saved perhaps from a subsequent nephritis. There is no reason why recent subjects of mild scarlet fever should not be allowed to get up at the end of ten days or a fortnight, provided that no complication has arisen, and care be taken to avoid chill or undue exertion. This more particularly applies to adult patients, who are often confined to bed for the regulation three weeks when it is quite unnecessary. In the case of children it is wiser to be on the safe side, and keep them in bed for a period of three weeks, and adults too, perhaps, in cold weather. Ordinarily, ten days or a fortnight of bed is quite sufficient for an adult. If the weather be dry, and the patient suitably clad, a moderate amount of outdoor exercise is to be recommended; but in damp or "muggy" weather young children should not be allowed to go out of doors unattended, as they are not unlikely to sit about and get chilled, and thus run considerable risk of contracting either renal or glandular complication.

As regards the clothing, linen should be worn next the skin during the febrile stage, but when the temperature has fallen flannel should be substituted, and either it or a woollen vest be worn throughout the period of convalescence.

Coming now to the treatment of diphtheria, although it must be admitted that no drugs are capable of exerting any direct influence over the course of the disease, we have a true specific in antitoxic serum. It is a great mistake when giving antitoxin to dispense with local treatment altogether. Some practitioners do not think it necessary to employ any local treatment if the patient has been injected with antitoxin. There is no doubt that if the fauces are in a state of inflammation, and are the seat of a deposit which is undergoing putrefaction, the condition is eminently favourable

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to septic absorption; and if by syringing out the fauces and nasal passages the parts can be kept thoroughly cleansed, such treatment is equally to be recommended, whether the case be treated by antitoxin or not. But in severe diphtheria you must be very careful not to unduly disturb your patient in the act of syringing, and to carefully avoid having anything in the shape of a struggle with him, because of the liability to heart failure in bad cases during the second week of the disease. Premising that every care is taken in respect to its employment, local treatment is certainly a factor which should not be ignored. The practice of treating diphtheria by means of powdered sulphur, with the idea, no doubt, of modifying the local process, is one which has been widely adopted. There is certainly no harm in blowing flowers of sulphur on to the surface of the throat, but at the same time it is very doubtful if any benefit results. However, this is but a personal opinion, and I would certainly not debar any one from using sulphur if desirous of trying it. Another form of treatment at one time very popular was to paint the exudation with some agent which was supposed to be capable of dissolving it *in situ*, such as lactic acid, papain, or lime water. I once tried an experiment with regard to this point: I put a large fragment of membrane taken from the throat into a saturated solution of lactic acid, and kept it there at a temperature as nearly as possible that of the body for twenty-four hours. At the end of that time there was no appreciable diminution in the bulk of the exudation. I was thoroughly convinced from this experiment that there was not much to be gained by painting the exudation with lactic acid. In cases for which for some reason antitoxin is not obtainable, the effect of staining the exudation *in situ* with one of the aniline dyes is worth a trial. That is a form of treatment which we were trying at the time when antitoxin was first introduced. By means of methylene blue in strong solution the faucial exudation was painted every few hours, with the result that it was darkly stained, and likewise the mucous membrane of the mouth and fauces. The urine and all the secretions became stained of a bright blue, and the milk even from a suckling breast became tinged with the pigment, so thoroughly did the methylene blue invade the system. Even the sheets and bedding were stained blue. We were certainly rather pleased with the results of this treatment until we got something better in the shape of antitoxin, and it was felt that there was something at any rate in the local treatment of diphtheria by means of the aniline dyes. Their germicidal effect on the diphtheria bacilli is evident from the intensity and rapidity with which these organisms became stained even at a considerable depth from the surface of the exudation.

As regards antitoxin, it is a true specific in diphtheria, and the results have fully substantiated the belief which was originally propounded as to its value, and a substantial basis was made out for our confidence in it before it was ever tried. It was known that certain animals were naturally immune against anthrax. It was found that if their blood were injected into other animals which were normally susceptible to anthrax, they could be thereby rendered immune to the disease. The next stage in the experiment was to render certain animals artificially immune, and then use their blood-serum as a protective agent for others. For the purpose of producing diphtheria antitoxin the horse was the animal chosen, because not only does it react well to diphtheria, but it is also a large beast, and therefore capable of yielding a correspondingly large amount of serum. As a result the poor old hack, which under less fortunate circumstances would have been consigned to destruction, was fed up and repeatedly injected with diphtheria toxin until its blood reached a certain antitoxic standard, and it was thereby rendered a most useful beast for the protection of mankind. And the results of the antitoxin treatment in practice have thoroughly justified the high hopes which were held as to its value. Antitoxin has been now in use some six or seven years, with the result that it has lowered the general mortality of diphtheria in the Metropolitan fever hospitals from a minimum of 30 per cent. down to about 12 per cent., and this year the mortality has been brought down even lower; in other words, the case mortality has been reduced to about a third of what it was before the introduction of antitoxin. In a certain group, viz. the laryngeal or croup cases, a previous mortality of 70 per cent. has been reduced to about 30 per cent. During the last three years at the South-Western Fever Hospital our recovery rate after

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tracheotomy from diphtheritic croup has been over 75 per cent., and this great reduction in the death-rate has been entirely owing to treatment by antitoxin. So that, instead of getting a tracheotomy death-rate of 70 to 80 per cent., we now get, as a result of the antitoxin treatment, a recovery rate of the same figure.

There is another class of case in which the immense value of the antitoxin treatment is well shown. I refer to cases of post-scarlatinal diphtheria. Originally post-scarlatinal diphtheria was the scourge of the Metropolitan Fever Hospitals. The mortality amongst children attacked with diphtheria when convalescent from scarlet fever was considerably over 50 per cent. Now that we have antitoxin to rely upon, this scourge has been practically wiped out of existence. We do not count on losing a child from post-scarlatinal diphtheria. One more benefit conferred by antitoxin is the obviation to a large extent of the necessity for tracheotomy. So much for the general results.

As regards the specific effects of antitoxin to be observed in detail, there are certainly four. One notices that the exudation separates more rapidly; the rhinorrhœa in bad cases of diphtheria is sooner checked, the swelling of the throat more speedily abates, and consequently the patient at an earlier stage of the attack gets into a condition of comparative comfort. A thing which is very noticeable, and, indeed, most striking, is the great difference in the general appearance of the diphtheria wards of the present day from what it used to be before antitoxin was employed. Instead of their containing a comparatively large number of patients with stertorous, impeded respiration, discharging noses, and swollen necks; now even the bad cases soon revert to a condition of relative comfort, and take an interest in their surroundings. Equally striking is the different view which the nurses now take of diphtheria work; in former days the nurses were often impelled to give up the work in consequence of their being unable to stand the constantly depressing effect of the diphtheria wards, so high was the mortality. At the present day it is entirely different.

In respect to the actual administration of antitoxin there are three points of importance I would like to mention. First of all, *give it early*. The paramount importance of commencing the treatment at an early stage of the disease cannot be too strongly insisted upon, statistical evidence upon this fact being overwhelming. At the Brook Hospital the results of treatment by antitoxin of 3000 consecutive cases during the years 1896 to 1899 inclusive were as follows:—the mortality of the cases treated on the first day of the attack was 0·0, on the second day 3·6 per cent., on the third day 6·7 per cent., on the fourth day 14·9 per cent., and on the fifth day and afterwards 21·2 per cent. I think it is hardly necessary to say anything more.

The second canon of administration is, *be sure that you give enough*. There has been a tendency to be somewhat afraid of antitoxin. I think the belief is founded on the erroneous idea that it is the antitoxic quality of the serum which is the source of possible danger. It is not so at all, however. It is the horse-serum in which the antitoxin is conveyed which is responsible for any ill effects. As a matter of fact, we find that in proportion as the serum is of high antitoxin potency, the after effects, which, as a matter of fact, are not serious, are as a rule reduced. I regard 4000 to 6000 units as a normal dose, and that is the amount I should give to start with, repeating it as often as might be necessary according to the severity and progress of the case. Until such time as the membrane is distinctly separating I would repeat the dose every twenty-four hours, or give it in half-doses every twelve hours. In a really bad case you may have to give four or five injections, or even more, to get the full benefit. Should the case be one either of extensive faucial involvement with nasal extension, or should there be distinct evidence of laryngeal implication, I would double the dose—*i. e.* give 8000 units to start with, instead of 4000. I do not think it is of any use to give larger doses than this. I notice that it is the teaching of some to give a very large dose—such as 16,000 to 20,000—to start with, and then no more; but I am quite convinced that this is a mistake. I should recommend spreading this amount over two to four days, according to the progress of the case. Certain of the sera obtainable at the present day are of considerable concentration, and a good serum will contain as much as 500 or 600 units in one cubic centimetre.

The third canon of administration is, *be sure that your serum is a reliable one*. There are

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certainly three taps of serum in the market which can be relied on—namely, that sold by Allen and Hanburys, which is prepared at the Jenner Institute of Preventive Medicine; secondly, that of Burroughs and Wellcome; and, thirdly, the serum sent out by the Parke Davis Co., either of which is perfectly reliable. The public Fever Hospitals are supplied with a special serum prepared at the laboratories of the Royal College of Physicians and Surgeons under the direction of Dr. Cartwright Wood. It should be remembered, too, that a certain amount of deterioration takes place in a serum if it be kept too long, and this deterioration is gradual and progressive. You may rely upon a serum keeping for three or four months, but after six months have elapsed, perhaps the best thing to do is to throw it away and get in a new lot.

As regards the actual injection, I can strongly recommend the use of the syringe called after M. Roux, at any rate until something better is produced. This is the one most generally used, and is a good practical syringe, for it can easily be taken to pieces and boiled, and therefore leaves nothing to be desired from the point of view of sterilisation. It is important that the syringe should be connected with the needle by means of a short piece of india-rubber tubing, because, if the needle be fixed on to the end of the syringe itself, it does not provide for the wriggling of the child, and is liable to get broken off in the skin. As regards the site of injection, some persons prefer injecting into the buttocks or under the skin of the abdomen; personally I think the best place is between the shoulders, for the areolar tissue in that region is loose and plentiful, and you can inject under the skin perhaps more easily there than in any other region. You must pinch the skin well up between the finger and thumb to enable you to more readily introduce the needle beneath it, and by injecting well under the skin the serum is more quickly dispersed. The amount of pain experienced varies very much in different patients, and you cannot anticipate the result of the injection in this respect. Some patients simply complain of the prick of the needle, whereas others will scream out with pain. I have in my mind an unfortunate medical practitioner into whom I injected antitoxin; he certainly was not enamoured of it, but said it was the most painful thing he had ever experienced. On the other hand, I have known children to sleep through their injection. Then, of course, it is of the highest importance to be careful as regards the sterility of the serum you are using, and of the syringe, both in the interests of the patient and of the reputation of antitoxin. Make a point of invariably boiling the syringe before using it; and in respect to your needles, I should advise keeping them in absolute alcohol. Directly you have finished using the syringe, run cold water through it so as to clear the serum out of it and the needle.

As regards the ill effects of serum injections, it cannot be denied that there are certain drawbacks, but they are not very serious.

The question of pain following the injection I have already referred to. About 50 per cent. of persons treated with adequate doses of antitoxic serum develop some form of rash during the course of the second week. This rash may take the form either of a morbilliform erythema or an urticaria; occasionally, however, it is scarlatinal. And this rash, according to statistics, occurs most commonly on the ninth or tenth day after the first injection. And in connection with the occurrence of a rash so regularly, the general practitioner will sometimes get a reputation for prophecy, which is not altogether to be despised, especially if he has told the patient beforehand that in about nine or ten days' time an eruption will probably appear. It certainly allays any unnecessary fear on the part of the patient.

The next most common after-effect is the occurrence of joint pains, but they are nothing like as common as the rash; about 5 or 6 per cent. would represent their incidence. These will last a few days and then clear up, and they may be attended with distinct arthritic effusion. Lastly, there is the possibility of an abscess. That is due to want of care on the part of the operator; at least it is said to be, but I am not quite sure that the statement is always justified. But by means of excessive care, however, you can reduce the number of abscesses to a very low percentage. As a matter of fact, during the course of last year at the South-Western Fever Hospital, out of some 600 cases of diphtheria, each case receiving on an average more than two injections, the total number of injections being considerably over 1200, we had only four abscesses. The rash, joint pains, and abscess are commonly attended with some rise

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of temperature, and occasionally there occurs a rise of temperature unaccompanied by any of these symptoms. This pyrexia is of unexplained origin.

Speaking generally as to the results of the antitoxin treatment of diphtheria, I can assert, after seven years' daily experience of it in practice, that it causes no ill effect worth mentioning in comparison with the enormous benefits which result from it. A certain amount of criticism, it is true, was levelled against the antitoxin treatment at its inception, but such criticism was founded either on ignorance or on sentiment. I remember reading an article in one of the monthly reviews written by a man not unknown in the literary field, who described his own experience of the treatment. He spoke very highly of the medical and nursing attention he received while an inmate of one of the Metropolitan Fever Hospitals, but his horror knew no bounds when, in addition to the pain and discomfort of diphtheria, his medical attendant actually proceeded to inject into him some "filthy horse-serum." He described the process with great wealth of detail and graphic illustration, and went so far as to assert his conviction that the material injected must have been horse-serum, because immediately after it was injected he "foamed at the mouth like a horse with the glanders."

In reply to certain questions which have been asked—first as to why antitoxin should not be injected into the arm as an equally suitable site as the back or the abdomen, I can only say that I have never tried it, but I see no reason why the injection should not be made there. The only objection which occurs to me at the moment is that under circumstances where a very large subcutaneous injection is required, the arm is rather a small limb to receive it—I mean a dose of from 10 to 20 c.c.

As regards the length of time after which the patient may be considered as free from infection, I can only say that one's attitude in this matter must depend upon what degree of authority one is inclined to ascribe to the result of a bacteriological examination. One would naturally like to rely upon a bacteriological examination, as it is so much more simple and satisfactory to have the answer reduced to terms of *plus* or *minus*. But looking to the fact that diphtheria bacilli are often found in the fauces of persons for many weeks after the attack is over, and that many cases have been published in which the bacilli have been present even for several months afterwards, it becomes impracticable to rely upon the bacteriological test for the purposes of isolation. In my own practice I am mainly influenced in faucial attacks by the length of time which has elapsed since the throat became clean. I allow my patients to mix with the community after the lapse of four weeks from the time when the exudation disappeared, and in support of this practice I would add that so far I have seen no reason to modify it in consequence of the occurrence of "return cases."

As regards the indiscriminate use of antitoxin in every case, no matter how mild, I have no hesitation in saying that it should not be given in every case. You would be guided by the likelihood of the attack developing into a severe one; in the case of adults it should be given, perhaps, more often than not, though in a large proportion it may safely be withheld; but in the case of young children, if I were convinced that the case were one of diphtheria, I would always give antitoxin, because of the possibility of a serious development in such patients.

As regards the use of a decoction of cinnamon in scarlet fever, I confess to never having given it since trying it in the cases I have mentioned to you, because the treatment is attended with certain drawbacks. It frequently induces vomiting, and as the dose is an ounce every hour—at least that was the dosage recommended by Dr. Carne Ross,—its administration constitutes a serious tax on the strength of certain patients; and, moreover, the complications of scarlet fever, if properly treated as they arise, for the most part are not very serious. So I have not felt disposed to subject any more patients to the inconvenience which attaches to its use.

In reference to the question of diagnosis between a case of diphtheria and one of septic sore throat, there is no doubt that great difficulty is experienced in some cases; and if the bacteriological examination should prove negative the diagnosis is rendered still more difficult. The question must, I take it, be then settled entirely by the general aspect of the case, and a consideration of those various clinical appearances to which I have referred.

As regards the supervention of paralysis, I have no hesitation in stating my conviction that if the

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paralysis supervene within a reasonable time after a throat attack, there being no other obvious cause for it, the case is almost certainly one of diphtheria. You will occasionally meet with cases in which the paralysis is so slight, that it is a matter of opinion whether or not it is present. Not infrequently a slightly nasal intonation may be the only evidence of paralysis available.

In conclusion I would like to say, in respect to the adoption of the antitoxic treatment, that we in the hospitals look to the general practioners to help us lower the mortality of diphtheria to an even greater degree than has been already attained. We have reduced the death-rate to nearly 10 per cent., but we cannot expect to get it much lower unless antitoxin be given earlier and somewhat more widely in general practice than it is at present, for in so many instances the patients are not received into hospital until a comparatively late stage of their attack.

Intra-Pelvic Drainage in Males.—Taylor has made dissections to determine whether the peritoneal pouch in the male pelvis can be drained to advantage similar to that of the female. He finds the rectum loosely attached behind, but more closely so in front, by the prostatic peritoneal aponeurosis, connected with the recto-vesical fascia. It, however, allows separation of the rectum from the prostate and bladder. The anterior wall of the rectum can be separated by blunt dissection and reflected toward the sacrum. Recalling the fact that the recto-vesical pouch is the most dependent point of the peritoneal sac, he reports a case where, through an unfortunate error in dressing, the patient was lost, though the operation was a success. A perineal incision was made, and the rectum separated by simply pushing the finger in the perineal wound up to the recto-vesical pouch—a blunt dissection with the finger. He thinks this plan, however, in spite of the misfortune in this case, has a decided advantage over any other plan when dependent pelvic and abdominal drainage is required. With the peritoneal cavity opened and assisted by one hand in the pelvis above, this procedure is as easy as opening the Douglas cul-de-sac through the vagina.—*Jour. A.M.A.*, December 13th, 1902.

PROGNOSIS OF TUBERCULOUS PERITONITIS IN CHILDREN.*

By G. A. SUTHERLAND, M.D.,

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THE prognosis in tuberculous peritonitis is a subject on which many and diverse views have been expressed. The great question as to whether the disease tends to run a favourable or an unfavourable course may be said to be still unsettled. Twenty-five years ago the prognosis was regarded as hopeless. Ten years ago it was considered more hopeful, especially if under surgical treatment. To-day we are still searching for more light, and although extensive statistics have been published on the Continent, which have considerably influenced opinion in this country, there are comparatively few published from English hospitals. I propose to limit my remarks on prognosis to the results of a study of forty-one cases of tuberculous peritonitis treated as in-patients at Paddington Green Children's Hospital. These represent all the cases of that affection which are available for statistical purposes from that hospital, a few having been rejected because the diagnosis was uncertain or the period under observation was too brief.

The first important point in the prognosis is to ascertain the proportion of recoveries to deaths. In these forty-one cases there are:

Recoveries	29	70·7 per cent.
Unrelieved	1	2·5 ,,
Deaths	11	26·8 ,,

If the results of medical treatment are compared with those of surgical treatment, the statistics are:

Medically treated, 27 cases.	Recovered	22	81·3 per cent.
	Unrelieved	1	3·7 ,,
	Died	4	15· ,,
Surgically treated, 14 cases.	Recovered	7	50 per cent.
	Died	7	50 ,,

In the case of a chronic affection like tuberculous peritonitis it can hardly be deemed correct to use the term "recovery" when the patient leaves the hospital or convalescent home—"apparently on the road to recovery" would be as far as one can safely go. The cases, therefore, have been followed up as well as possible after leaving the hospital,

* Read before Society for Study of Disease in Children.

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and the statistical results, both favourable and fatal, are drawn up after keeping the patients under observation for the longest possible time. The cases which are entered as "recovered" were under observation for the following periods:

Under 3 months . . .	2
From 3— 6 months . . .	7
„ 6—12 „ . . .	1
„ 1—2 years . . .	4
„ 2—3 „ . . .	4
„ 3—4 „ . . .	1
„ 4—5 „ . . .	4
„ 5—6 „ . . .	3
Over 6 years	3
Total . .	29

Considering the number of cases which were under observation for a year or longer—namely 19 or 65·5 per cent.—it will perhaps be admitted that the term "recovery" is justified. Further, in those cases the signs of active disease had ceased, the traces of local disease became less evident with each succeeding year they were observed, and the general strength seemed steadily to improve.

The Natural Course of Tuberculous Peritonitis.—Tuberculous peritonitis in its uncomplicated form is an affection which lasts usually for from six months to a year, or longer. The natural course of the disease in a fairly healthy child would appear to be towards ultimate recovery. Perhaps in no form of tuberculosis does nature make greater or more successful efforts to cope with the disease. The formation of fibrous tissue is nature's method of shutting up the tubercles, cutting off their supply of nourishment, and starving them out. When their vitality is gone and all active peritoneal tuberculosis has ceased, nature next directs the powers of the body to the absorption of the dead tubercles and the fibrous tissue surrounding them. Sometimes the effort is completely successful, sometimes the absorption is only partially complete, and sometimes complications arise from outside causes which put an end to the process. If this process of fibrosis is to be regarded as the natural course of the illness towards a favourable issue, the prognosis will depend on the patient's powers of furthering it. Consequently a strong family history of tuberculosis, or an infancy passed under bad hygienic and dietetic conditions, or a constitution of slight resistent power, or a history of one or more severe attacks of infective fever during early life, must be regarded as a factor which influences unfavourably the prognosis in any given case.

The Prognosis in Uncomplicated Tuberculous Peritonitis.—The disease presents itself under various clinical aspects. There may be an acute onset, with typhoid-like symptoms, followed by a very severe illness, characterised by ascites, abdominal distension, diarrhœa or constipation, and high temperature. Although one would hesitate to speak decidedly during the acute stage of the illness, it is not uncommon for such cases to terminate in complete recovery. Most of the cases begin insidiously. The ascitic type, in which fluid is poured out more or less freely, owing to the irritation produced by many scattered tubercles, is one in which the prognosis is favourable if it is succeeded by a fibro-plastic formation, which is the natural termination. Those cases in which the fibro-plastic formation is active from the start do well as a rule. Cases of tumour formation, rolled-up omentum, or puckered-up mesentery, or matted intestine, may present no active symptoms, are extremely chronic, and usually terminate favourably. A common clinical type is that in which tuberculous pleurisy is an associated condition, and the prognosis does not appear to be unfavourably influenced by this association.

Various symptoms in the course of the disease affect the prognosis. A favourable condition may be said to be present when the *temperature* is subnormal, or normal, or only slightly raised; and even when there is a considerable evening rise to 102° or 103°, it is wonderful how well and how long the patient will hold his own, provided there is a daily remission and apyrexial period. On the other hand, continuous pyrexia, even of moderate degree, is unfavourable, is usually accompanied by progressive loss of strength, and is suggestive of some complication. A continuously rapid *pulse*, over 110 per minute, is to be regarded as indicative of acute abdominal disease, and is of unfavourable prognostic significance.

Diarrhœa when prologed is of bad nomen, and suggests one of the gravest complications, tuberculous ulceration of the bowel. Great *distension of the stomach and intestines*, when paralytic in origin, is of unfavourable significance; when it is obstructive in origin it may be amenable to surgical

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treatment. *Rapid wasting* must be looked on as an indication that the disease has gained the upper hand locally, or is becoming generalised. The defensive powers of the constitution are invariably weakened in the presence of rapid emaciation. *Recurrent attacks*, with or without marked pyrexia, affect the prognosis unfavourably. They tend to exhaust the resistent powers of the patient, and they point to fresh infection from an active, although possibly unrecognised, source.

The Prognosis in Complicated Tuberculous Peritonitis.—The complications which may precede or arise from tuberculous peritonitis seriously affect the prognosis, and one or more of them will usually be found to be present in the fatal cases.

1. Tuberculous ulceration of the intestine. This may be small in extent and not associated with any marked symptoms. When it is extensive, as indicated by intractable diarrhœa and the passage of blood and mucus, the prognosis is unfavourable.

2. If extensive caseation of the mesenteric glands or of tuberculous masses exists, recovery is delayed, fresh infection of the peritoneum is apt to occur, and the risk of abscess formation and rupture is constantly present.

3. Localised suppuration, from infection through glands or the intestine, is a complication which, if unrelieved by surgical treatment, is apt to retard recovery and lead to still more serious complications.

4. Obstructive symptoms from intestinal matting, or the pressure of tuberculous growths, are, as a rule, not marked. If progressive and unrelieved the prognosis is bad, but surgical interference may prove successful in removing all imminent danger.

5. In the presence of pulmonary tuberculosis, with definite physical signs, the prognosis is unfavourable. It must be remembered, however, that dulness and crepitations are frequently present at one or both bases, owing to the diaphragm being pushed upwards, the lower portions of the lungs being thrown out of action, and congestion occurring in consequence. With the relief of the abdominal distension, these pulmonary signs will usually disappear entirely. The prognosis is very bad in the case of the following complications:

6. The rupture of a glandular or other abscess, or the perforation of an intestinal ulcer into the general peritoneal cavity.

7. The onset of tuberculous meningitis.

8. The occurrence of general miliary tuberculosis.

As confirming the serious prognostic influence of the various complications, the causes of death in the fatal cases may be given here. Six of the eleven cases were examined post-mortem:

	Cases.
Purulent peritonitis from intestinal perforation or ruptured abscess	5
Intractable diarrhœa (? intestinal ulceration)	2
With cerebral symptoms (? tuberculous meningitis)	2
Tuberculosis of peritoneum, pleura and lung; with partial intestinal obstruction by a band	1
Cause unknown	1
Total	11

The Prognosis as affected by Medical Treatment.—The results of medical treatment as given above may be regarded as favourable. If the prognosis in uncomplicated tuberculous peritonitis depends on improving as much as possible the patient's resistent powers until the tuberculous process is checked, and increasing his aggressive powers until the tuberculous products have been entirely removed, it is plain that medical treatment directed to these ends will be of the highest importance in making the prognosis more favourable. In the complicated forms of the disease medical treatment is of less direct value.

The Prognosis as affected by Surgical Treatment.—In the complicated forms of tuberculous peritonitis the prognosis is much more serious, and here it is that the prospect may be entirely changed by surgical interference. One striking case is before me, that of a girl of three years, on whom Mr. Watson Cheyne performed laparotomy for obstruction and short-circuited the small intestine. The patient made an excellent recovery. In another case, with suppurative peritonitis from a perforation of the cæcum, a fatal termination ensued.

As regards the surgical treatment by simple laparotomy — the so-called cure for tuberculous peritonitis—our results are not good, namely six recoveries and six deaths. It is not suggested that the fatal results were due to the laparotomy, for the deaths occurred at periods of from one to six months after the operation. But it is clear that the operation was ineffectual in preventing the complications which led to a fatal termination. The cases which recovered may be said to have

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done so in spite of the operation, for they presented in their post-operative stages no striking changes and no clinical developments which could not be equally well observed in the cases treated medically. At one time I was a supporter of laparotomy as a routine treatment, but further experience has altered my opinion. A girl of six years, the subject of fibro-ascitic tuberculous peritonitis, was considered a suitable case for laparotomy, but operation was deferred for unavoidable reasons, and she was sent to the country. While she was there the abdominal contents became harder and denser until they felt like plaster of Paris, the fluid disappeared, and the girl improved. Some months later she was so much better that operation was not considered necessary, and during the next year there took place a gradual absorption of all the dense material, until finally she was quite restored to health and the abdomen to a normal condition. In other cases before me—cases of acute tuberculous peritonitis, of tumour formation, of the ascitic type, and of the fibrous variety—I find that as good results may follow from medical as from surgical treatment, and I do not find that the course of the disease is appreciably affected by laparotomy. From the post-mortem evidence also it is plain that, whether the case was medically or surgically treated, death was not due to uncomplicated tuberculous peritonitis, but to some complication beyond relief either by medical measures or by laparotomy.

The conclusions drawn above may be summarised as follows:

1. In uncomplicated tuberculous peritonitis the prognosis is good.

2. When tuberculous pleurisy is present the prognosis is still favourable.

3. The prognosis is rendered less favourable in the case of (*a*) a strong family history of tuberculosis, (*b*) an infancy passed under bad hygienic and dietetic conditions, (*c*) a constitution of feeble resistent power, or (*d*) a history of severe infective illness in early life.

4. The prognosis is rendered less favourable in the presence of one or more of the following symptoms:—continuous pyrexia, rapid wasting, persistent diarrhœa, rapid pulse, and recurrent acute exacerbations.

5. The prognosis is rendered less favourable in the presence of one or more of the following local complications:—(*a*) tuberculous ulceration of the bowel, (*b*) extensive caseation of the mesenteric glands or of tuberculous masses, (*c*) localised suppuration from infection through glands or the intestine, and (*d*) obstructive symptoms from bands or matting of the intestine.

6. The prognosis is bad in the case of the following complications:—(*a*) the rupture of a suppurating gland or the perforation of an intestinal ulcer into the peritoneal cavity, (*b*) pulmonary tuberculosis, (*c*) tuberculous meningitis, and (*d*) general miliary tuberculosis.

7. In tuberculous peritonitis the prognosis is not appreciably affected by simple laparotomy.

Fatal Acetonæmia following an Operation for Acute Appendicitis.—The patient of G. E. Brewer was a schoolboy of twelve years. A perforated gangrenous appendix was found in a small abscess cavity containing two drachms of foul pus. Duration of anæsthesia was twenty-five minutes. On the third night after operation he suddenly awoke and began to scream. These attacks were renewed at intervals during the next day. The wound conditions appeared normal, and there was no evidence of cranial nerve palsy or irritation. No cause for the peculiar mental condition could be discovered, but a peculiar sweetish, ethereal odour of the breath was noticed, and a diagnosis was made of acute auto-intoxication from the presence of acetone and diacetic acid in the blood. This conjecture was confirmed by finding large quantities of both substances in the blood and urine. Free catharsis, diuresis, and intra-venous injections of chemically pure sodium bicarbonate were instituted, but the patient died about twenty-four hours later. In the final hours the mental symptoms assumed more of a passive type. Brewer has never known of a similar case. Possible causes are trauma, sepsis, drug action, and auto-intoxication (ptomaines, uræmia, and acetonæmia). As regards the possible relation of anæsthesia to the condition, Brewer notes the presence in the urine of a pathological amount of acetone in seven out of thirty-three cases following anæsthesia, the records being taken from his hospital service.—*Medical Record*, December 6th, 1902.

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A CLINICAL LECTURE ON MYOPATHY.

Delivered at the Hospital for Sick Children, Great Ormond Street, London, November 6th, 1902.

By F. E. BATTEN, M.A., M.D., F.R.C.P.,

Assistant Physician to the Hospital and to the National Hospital for Paralysed and Epileptic, Queen Square.

GENTLEMEN,—I have chosen for the subject of my lecture to-day a group of cases which are collectively known under the title "Myopathy." It is unnecessary for me here to go into the history of the disease which has gradually been built up during the last fifty years.

The characters which the various groups of this form of muscular atrophy possess in common are —Slow progressive loss of power in muscles or groups of muscles, associated with atrophy or hypertrophy of the affected muscles, absence of all fibrillary contraction, and absence of any qualitative electrical change.

The atrophied muscles do not in their grouping correspond to an affection of the spinal cord or of nerve, such as is seen in progressive muscular atrophy of a myelopathic origin. There is a marked tendency for the disease to be hereditary and to affect various members of a family. The muscles for the movement of the eyes and the muscles of the larynx, pharynx, and diaphragm are never affected.

Cases of myopathy may be divided into groups, each of which has certain characteristics, but although it may be convenient for the purpose of description to divide the cases into such groups it must not be supposed that all cases will conform in every detail to the characteristics of a group, and it is possible to find transitional cases which possess characters of two or more groups. I hope to-day to be able to illustrate each group by a typical case of the disease, and the following are the types which will be described:

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(1) The infantile type.

(2) The facio-scapulo-humeral type (Landouzy-Dejerine).

(3) The juvenile type—scapulo-humeral (Erb).

(4) The pseudo-hypertrophic type.

In close relationship with these, but possessing a somewhat different pathology, is—

(5) The peroneal type (Charcot, Marie, Tooth).

The infantile type.—The special features of this type are—that the disease commences in infancy; the babe does not learn to sit up at the usual age; even when this is learnt the child has considerable difficulty in maintaining the erect position. The child may at three to four years old learn to walk, but more often these children never learn to walk. They are usually very intelligent, learn to talk during the first and second years, and are very contented children.

If at a later age the child is placed on its back and asked to sit up, it turns over on to its face, draws up the legs, and then by a process of pushing with the hands raises the trunk into the erect position; the legs are then manipulated by the hands so as to bring them in front of the trunk.

All muscles of the body are very small, but there is no movement which cannot be performed; there is no localised atrophy of any muscle or portion of a muscle, nor is there hypertrophy of any muscle. Sensation is perfectly normal in these children and the superficial reflexes usually present. The knee-jerks and other tendon reflexes are diminished or absent according to the degrees to which the atrophy has progressed. If these children be lifted up by the shoulder they have the characteristic feature which is felt in nearly every type, namely, the loose condition of the shoulder-joint. With regard to the electrical reactions, they show a greatly diminished reaction to faradism and to galvanism. One point is of great interest with regard to the faradic current, viz. that these children will bear without any appearance of pain or discomfort a current which is so strong that it cannot be borne by the normal adult—and yet to ordinary forms of stimuli and to galvanism the children are as sensitive as the normal individual. The condition will, however, be best realised by a careful study of the following case.

D. L—, a boy æt. 4½ years, is the third child of a family of four. His father and mother are healthy and well, and as far as can be ascertained no such weakness as this boy presents has occurred in the family. The boy has never been able to stand or walk; he can sit up, but cannot kneel. He talks well and is very intelligent. He swallows food well but is rather slow in doing so. The arms are moved in all directions, and the boy can use them well, but the guardian says "they turn all round." He is clean in his habits. He is very thin and small for his age, though not very short. He is unable to walk and unable to stand, and if an attempt be made to place him on his feet he flops down in a mass on the floor. While holding the boy by the shoulders he has that feature which is so characteristic of the group of myopathies, viz. the feeling as if the child would slip from one's hand. In order to get across the room the child lies on the floor and rolls round and round on his long axis, and having arrived at the point desired he goes through the following manœuvre to get into a sitting position:—With his face to the floor he draws his legs up under him into a flexed position; he then by pushing with his hands raises the trunk into an erect position, so that he comes to sit on his heels, and the hypotonia of the muscles is such that the femora and the tibiæ lie practically parallel to one another. While thus kneeling the arms support the weight of the trunk. The legs are now manipulated so that they are brought round in front of the trunk, and the child gets into the sitting position. This position is not, however, one of stable equilibrium, and the child is most easily upset, and in order to get again into the sitting position has to repeat the manœuvres above described.

The muscular development of the child is very small, but there is no localised atrophy either of muscles or portions of muscles, nor is there any hypertrophy. All movements can be performed with the arms, and there is no winging of the scapulæ. The grasp of the hand is feeble, but the child can oppose the thumb and little finger readily. The child is unable to extend the leg on the thigh owing to weakness of the quadriceps extensor. The weakness of the psoas and iliacus, and of the glutei is also considerable. The extensor and flexors of the feet are fairly good, and the toes can be moved naturally. Both the abdominal muscles and muscles of the back are weak, but

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still have sufficient power to flex the trunk and extend the spine.

The spine is straight, but there is at the lower part a small depression which might be the remnant of an occult spina bifida. The bones of the hands are extremely small, as well as the bones of the limbs. The feet appear long in comparison to the size of the legs, and have the same minute appearance as the hands.

All movements of the face, both voluntary and emotional, are good; and movement of the eyes, tongue, and palate are all natural.

With regard to tactile sensation, heat, and cold, and painful impression are readily appreciated; but the child exhibits a curious indifference to a strong faradic current, and even to so strong a current that it cannot be borne by a normal adult. The muscles when tested with faradism react to a very strong current, but the strength is such that it produces a most painful contraction in the normal adult, and even this current will produce no good contraction in the quadriceps extensor or glutei. The muscles react to galvanism, but require a strong current, the KCC being greater than the ACC. The boy does not exhibit the same tolerance to this current which he exhibits to the faradic stimulation.

The knee-jerks are absent, and the plantars exhibit a normal flexor response.

This peculiarity with regard to the insensibility to faradic stimulation has been noticed under certain conditions other than myopathy; for instance, it was present in a case reported by Bruce and Thomson of progressive muscular atrophy following a spinal lesion in a child. Again, it is found also in infantile paralysis where there is extensive affection of muscles. The painful sensation caused by faradism is partly due to skin stimulation and partly due to active muscular contraction, the painful impression being derived from the sensory nerve termination in the muscle spindle and tendon organs.

Now if the muscle-fibres of a muscle are so atrophied that little or no response is obtained in them by faradic stimulation, then no pressure will be exercised on the sheath of the muscle spindle, and hence no pain will be produced. Such an explanation does not, however, explain the almost complete absence of pain from skin stimulation.

The problem is an interesting one, for a complete explanation of the phenomenon has not been obtained.

Passing now to the second type, *the facio-scapulo-humeral or Landouzy-Dejerine type*, let us consider the special feature of this group. The onset of the disease is subject to great variation, in some cases the first symptoms being observable in earliest childhood, while in others the disease does not manifest itself until adult life in the third or fourth decade. The most usual period for the onset of disease is during the second decade of life. The disease manifests a marked hereditary tendency, and affects both sexes alike, being transmitted by the male or female to the male or female with like frequency.

The leading features of the disease are as follows:—An almost expressionless face, with a wide, long-drawn-out mouth, usually held somewhat open. The eyes cannot be fully closed, and the brow cannot be raised, the cheeks cannot be blown out, and the patient cannot whistle. The movements of the eyes, tongue, masseters, and palate are normally performed. With regard to the limbs and trunk there is marked wasting about the shoulder and upper arm muscles, and also about the gluteal and thigh muscles, while the muscles of the forearm and legs below the knee may be well developed, giving to the patient an appearance which is very characteristic of the disease. There is no fibrillary tremor, the sphincters are unaffected, and the reflexes are only lost when the atrophy becomes extreme. The muscles do not exhibit the reaction of degeneration. The condition is well illustrated by the following case:

E. S—, æt. 8 years, is the eldest of a family of three boys. According to his mother's statement he has never been able to close the eyes, and as quite a baby slept with them wide open. When two years old he was taken to Moorfields Hospital in consequence of tears falling over his cheeks, and for relief of the condition the canaliculi were slit up. At Christmas-time when about two years old he was presented with a trumpet, which he was unable to blow until the mother held the corners of his mouth. The boy was first brought to me about three years ago, and his face presented the lack of expression which is so characteristic of the disease. There was marked weakness of the frontalis, the orbicularis palpebrarum and oris,

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and weakness of the zygomatici. The boy was unable to close his eyes completely and was unable to whistle. All movements of the eyeball were well performed. With regard to the trunk muscles, although he was thin he could perform all movements of the arm well; there was, however, some weakness of the anterior tibial and peroneal muscles of the legs, and this has increased during the past few years. He could get upstairs without much difficulty, and could raise himself from the floor without performing any unusual manœuvres. The knee-jerks were present and equal. There was no fibrillary tremor of the muscles, and no affection of bladder or rectum, or alteration in sensation. No reaction could be obtained in the muscles of the face to faradisation, and the anterior tibial and peroneal muscles did not react readily.

The third type of myopathy is that known as the *juvenile type of Erb*—the scapulo-humeral type,—and by many people this is regarded simply as a variety of the former, the facio-scapulo-humeral type. But while fully recognising the probable identity of the affection, it seems well to classify it as a separate type because of the absence of affection of the face, the occasional occurrence of hypertrophy, and the age of incidence—namely, during the second decade of life and not from birth.

The disease affects families and both sexes alike. The following case, although possibly not perfectly typical of the type, yet exhibits most of the characteristic features of the group:

W. F—, æt. 7 years, learnt to walk when two and a half years old. He frequently fell down, and was unable to walk upstairs. He is the first of two children, the other being a girl. There is no family history of any similar complaint. The child is generally thin, and shows marked wasting about the scapula and upper arm muscles in both upper extremities. There is marked weakness of the serratus magnus, but the lower portion of the pectoralis major is fairly developed. The trapezius is atrophied and weak. The left sterno-mastoid is absent, and the right is very thin.

With regard to the legs, the thighs are wasted, while the legs below the knees are fairly developed. He is able to raise himself from the ground without difficulty, but has considerable difficulty in going upstairs. The knee-jerks are present.

The fourth type is that which, owing to its more frequent occurrence, is best known—namely, *the pseudo-hypertrophic type*. This disease has certain well-marked characteristics. Unlike the other forms of myopathy, this type tends to affect the male sex far more frequently than the female—but the disease is usually transmitted through the mother and not the father; the mother having brothers who were similarly affected, although she herself was unaffected.

Several members of a family may be affected, and it is not uncommon to see somewhat different varieties of this disease in the same family. For instance, one boy will have the large calves, large glutei, and large infra-spinati muscles, and the atrophied lower portion of the pectoralis major; while another boy will show no hypertrophy of muscles, but only atrophy, the atrophy having the distribution characteristic of the type.

The disease commonly manifests itself between the ages of five and six, but cases have been recorded in which the onset of the disease has been in the third and fourth decade of life.

The symptom which first attracts the attention of the parent is that the child frequently falls and is unable to get up from the ground without help. Sometimes the difficulty with which the boy goes upstairs is the symptom which first attracts attention. The size of the calves may have been noticed, but is usually regarded as a sign of strength rather than one of weakness.

The gait of these patients is very characteristic: they stand with the shoulders thrown back, the head erect, with the abdomen protuberant, and a marked lordosis. Owing to the hypertrophy of the glutei the buttocks appear very prominent. When the patient begins to walk the gait is waddling, and the weight of the trunk is swung vertically over the head of the one femur while the other leg is swung forward. All goes well so long as no obstacle is met with, but then, as there is very considerable difficulty in raising the foot off the ground owing to the weakness of the flexors of the thigh, the patient is unable to raise the foot over the obstacle. So, also, when the patient attempts to go upstairs the one leg is swung out laterally on to the stair above, the hand is then placed on the knee and the weight of the trunk is thrown forward, the knee is straightened and the step accomplished, and the same manœuvre is repeated

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for the next step. It is only in the early stages of the disease that mounting steps is possible.

When placed on the floor, the child lying on its back, and asked to stand up, the patient goes through a regular set of movements. The patient first rolls over on his long axis so that the face is turned to the floor; the knees are now flexed, and a kneeling position is assumed; then the knees are extended, and the body is supported on four points forming a pyramid—the apex being the buttocks, and the base the four extremities. The feet are now brought nearer to the hands, and then the hands are placed firstly on the shins, then on the knees, and in this way the trunk is raised into the erect position.

Certain muscles are found atrophied, certain muscles hypertrophied. The muscles most commonly atrophied are the biceps, the latissimus dorsi, the serratus, the lower portion of the pectoralis major, the flexors of the thigh, and the extensors of the leg. The muscles most commonly hypertrophied are the gastrocnemii, the glutei, the deltoid, triceps, and infra-spinatus. The atrophy of the lower portion of the pectoralis major and the hypertrophy of the infra-spinati are possibly the two most constant features of this disease.

The electrical excitability of the muscles is always diminished, but there is no reaction of degeneration. The presence or absence of the knee-jerks depends on the condition of the quadriceps extensor, and more especially on the vastus internus, as was shown by Sherrington. When this muscle is atrophied the knee-jerk is lost.

The last case which I show this afternoon has some of the features of a myopathy, but has also certain features which separate it from that group. This type is known as the *peroneal type* of muscular atrophy (Charcot, Marie, Tooth). The characteristic features of this disease are its markedly hereditary tendency—the disease passing from father or mother to daughter or son indiscriminately. The hereditary tendency has been traced through five generations. The disease usually manifests itself before ten years of age, but many cases also commence during the second decade of life. The atrophy begins in the peroneal muscles of the legs and affects the more distal portions of the muscles before those more proximately situated. There are marked vaso-motor disturbances, and some alteration of sensation is often present; the disease is of slow progress, and after the legs have become affected the small muscles of the hands begin to suffer.

The following case illustrates the various features of the disease:

F. S—, æt. 9 years, had measles when three years old, with bronchitis and pneumonia. He was very ill for some time, and on recovering his mother noticed that he dragged the left leg. He complained of no pain. About seven months later the right leg became weak, but the boy could always get about. When four years old he was put into irons, and these he wore for eight weeks, when, owing to frequent fracture, they were discarded.

When six years old he began to lose the power in his hands, and he was unable to lift objects with his fingers. The wasting of the limbs is slowly progressive. He is the second of five children, all the others being healthy, and there is no history of any similar complaint. The boy is rather pale and thin, but presents no abnormality of the head, eye, or face muscles. The tongue and palate move well, and he can whistle.

The movements of the upper and fore arms are well performed, but there is a slight tendency to drop-wrist in both hands. The grasp is fairly good. There is wasting of the thenar and hypothenar muscles of both hands, and also of the interossei. The boy is unable to oppose the thumb and little finger, and although adduction and abduction of the thumb can be performed, both are very weak; both hands are in the typical "claw" position, and the weakness of the interossei is very marked. The scapular, pectoral, and shoulder muscles are well developed. The boy walks with some difficulty, lifting the leg high in order to prevent the toes from dragging on the ground. If placed on the floor and asked to get up, he does so with ease, and without any such manœuvre as is seen in other forms of muscular atrophy. The feet are in a position of equino-varus, and the boy is unable either to dorsiflex or rotate the foot outward.

There is very marked atrophy of all the muscles below the knees, so that the legs are mere sticks. The thighs are bottle-shaped, owing to the atrophy of the distal portion of the muscle, although this feature is not so marked at the present time as it was two years ago, owing to the progress of the atrophy up the thighs.

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There is no alteration of sensation to either heat or cold, touch or pain. The knee-jerks are present, and the plantar reflex gives a flexor response. With regard to the electrical reactions, the unaffected muscles react normally, whilst in the affected muscles no reaction can be obtained to faradic stimulation; there is, however, no reaction of degeneration.

I have endeavoured this afternoon to put before you types of muscular atrophy or hypertrophy which, so far as our present knowledge goes, are not due to an affection of the spinal cord or nerves, but are due to profound alteration in the muscles themselves. But, although I have for the purpose of this lecture separated the various forms of myopathy into four groups, viz. the infantile, the juvenile, the facio-scapulo-humeral, and the pseudo-hypertrophic, it must be remembered that although the majority of cases can be placed in one or other of these divisions, there occur cases which are intermediate between the various types, and no lecture on the subject would be complete without the recognition of this fact.

Amputation for Blood Poisoning.—Dörfler has expressed the opinion that amputation is of no avail in saving life in cases of phlegmonous inflammation of the extremities in which bacteria have gained access to the general vascular circulation. Wolff (quoted in 'Centralblatt für Chirurgie,' January 25th, 1902) strongly opposes this statement, and upholds his own views by citing cases of phlegmonous inflammation, malignant œdema, and moist gangrene which were observed in Bergman's clinic. Amputation was strictly reserved as an operation of last resort in this clinic. In the cases in which amputations were performed, the clinical symptoms of a general intoxication were pronounced, and the presence of bacteria in the blood was demonstrated, yet the operations resulted in the saving of life. The reported histories of the patients demonstrate beyond doubt that some of them would have succumbed but for the performance of an amputation.—*Therapeutic Gazette*, December, 1902.

THE EFFECT OF PREGNANCY ON CHRONIC HEART DISEASE.*

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(*Continued from page* 182.)

I SHALL now consider the effect of pregnancy on various cardiac conditions.

Many persons suffering from heart disease pass through pregnancy, parturition, and puerperium without any manifestations of failing compensation. In some exceptional cases I believe that one or two pregnancies may not shorten the patient's life, but in the majority of instances pregnancy hastens cardiac failure. There are various factors that influence the prognosis, and each case must be judged on its own merits.

1st. The Position in Life of the Patient.—This is frequently the determining cause of the heart failure, for women who have to do hard work on insufficient food do not live through many pregnancies. The imperfectly nourished heart-muscle, having too little rest, and being often liable to be overstrained, speedily becomes degenerated. The effect of work on aortic and mitral cases is different. I have often observed that patients with aortic regurgitation may do hard work right up to the eighth month or so, while in mitral disease the exertion of going up stairs three or four times a day has been sufficient to break down compensation at the fifth or sixth month. When a patient is more fortunately situated she has a better chance of maintaining compensation, for she is able to save her heart unnecessary strain and to take such measures as may be required to improve her general health.

2nd. The Condition of the Heart-muscle.—This is the most important question of all, but unfortunately it is often an unknown quantity. When a patient with valvular disease, who has never done any hard work, is pregnant for the first time, it is very difficult to tell how the heart will behave, and we must carefully watch the course of events. If, however, there has been a previous pregnancy,

* Presidential Address to the Chelsea Clinical Society on October 28th.

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we have some ground to go upon, and are able to form a more reliable prognosis. Presuming that a patient starts child-bearing with a healthy heart-muscle, the gradual onset of failure may be taken to be the equivalent of the commencement of degeneration, the special influence that brings this about being an impoverished and impure condition of blood. Under the ordinary circumstances of heart disease the lungs are largely responsible for the impurity, but in pregnancy, besides there being a normal deficiency of hæmoglobin, the blood may become toxic, owing to the failure of the kidneys to eliminate fœtal waste products. Constipation is another cause of impurity, owing to the absorption of retained fæcal matters. Prolonged lactation with anæmia is yet another cause of degeneration of heart-muscle, and it is unsafe for a patient with valvular disease to become pregnant until the health is improved. The introduction of specific poisons from without may also cause this degeneration. I have a patient with double mitral disease at present under treatment, who was confined in November, 1900, with her first child, everything being natural throughout. Early in March, 1901, she had diphtheria, and miscarried at four months, three weeks afterwards. Compensation failed, and she is still quite unable to undergo any exertion on account of breathlessness and palpitation. This also applies to several other diseases.

3rd. The Situation and Extent of the Disease.—All are agreed that pregnancy materially alters the relative duration of life in chronic valvular disease, and that mitral stenosis has by far the highest rate of mortality. There, however, all agreement ends, for great differences of opinion exist as to the absolute death-rate of this disease, and no decision has been arrived at as to the relative danger of the other forms. This is chiefly owing to the different aspects from which the obstetric and the general physician view the subject, for each being guided by his own experience arrives at a different conclusion. The former sees all the worst cases of heart failure and only a portion of the compensated ones, whereas the latter sees only a few cases during parturition, but has a larger experience of those that have managed to live through a varying number of confinements; these are therefore exceptional cases, and represent the survival of the fittest. My own experience leads me to place the order of relative danger thus :—Mitral insufficiency, aortic insufficiency, aortic stenosis, mitral stenosis, aortic and mitral insufficiency and aortic insufficiency and mitral stenosis combined; mitral insufficiency being, in my opinion, the least dangerous disease in the pregnant as well as the non-pregnant. The prognosis must be very guarded when the endocarditis has attacked more than one orifice at the same time; for, apart from the double lesion, the myocardium is more likely to have been affected than in a less extensive inflammation, and a small amount of fatty degeneration will then produce alarming results. Of course this does not always occur, as will be seen subsequently, but the possibility must be borne in mind.

4th. Previous Pregnancies.—Failure will, as a rule, occur earlier in each successive pregnancy, but a quick succession of pregnancies hastens it, the heart having no time to recover itself.

Gravido-cardiac symptoms average themselves, as it were, into two distinct types, the mitral and the aortic. When more than one orifice is involved the predominant type depends on the valves that have primarily received the greatest amount of injury.

The mitral type presents many special features, and as we have them exemplified in mitral stenosis I shall take that disease for my text; for regurgitation, whether primary or secondary, presents the same symptoms, although in a lesser degree. The mortality of stenosis when complicated by pregnancy ranges, according to obstetricians, from 30 to 60 per cent., the majority considering that 50 per cent. is probably about the average proportion of deaths. Against this I find from my own notes that fifteen women have had seventy-seven pregnancies between them, sixteen of which have resulted in miscarriage; only one of the fifteen died (tenth day); the rest survived puerperium, and were able to get about afterwards. I do not attach the slightest importance to these figures from a statistical point of view, but they form a dot in the foreground and take off the severity of the obstetrician's heavy mortality.

In well-compensated mitral stenosis the patient may pass through pregnancy without any signs of heart failure at all, there being but little more dyspnœa on exertion during the later weeks than under ordinary circumstances. Some cyanosis may

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be present during the second stage of labour, but it soon passes off after delivery. There is no tendency to flooding, and the patient quickly rallies. When the disease is further advanced and the mitral orifice more constricted, the heart-muscle may be unable to carry on the work effectively, and signs of failure show themselves during the later months. The lungs are almost invariably the first organs to suffer, pregnancy increasing the high tension already present in the pulmonary vessels. Palpitation, dyspnœa, cough, and in some cases hæmoptysis, are the usual symptoms complained of, bronchial catarrh and œdema of the bases being generally found on physical examination. Bronchial catarrh and broncho-pneumonia may be present while the heart is still compensated, but œdema of the base may, under these circumstances, be taken as a sure indication of failing compensation. These lung complications increase the tension in the right ventricle and the venous system generally. The functions of the other organs of the body are thus impaired, and add materially to the gravity of the case. During the first stage of labour the breathing may become embarrassed and the pulse irregular and rapid if there is much straining. In the second stage the face becomes extremely cyanosed, the breathing proportionally embarrassed, and the pulse uncountable during the pains, but immediate relief follows delivery when the heart-muscle is sound. Great care, however, is necessary, even in the most favourable cases, until the various organs recover from their congested condition.

Unfortunately this is only one side of the picture; another is seen when kidney, as well as lung complications, are present, and the heart-muscle is degenerated. Under these conditions the lung symptoms are liable to assume a much more serious aspect; broncho-pneumonia may occur in patches, and pulmonary embolism is not a very rare event. When small, emboli may only give rise to temporary dyspnœa and syncope, but when larger arteries are involved fatal results may ensue. It may follow some apparently trivial movement, or there may be no apparent exciting cause. The patient suddenly becomes cold and clammy, her aspect assumes a death-like pallor, the lips being slate-coloured. There is intense dyspnœa and fighting for breath, the pulse becomes uncountable, and death may take place in spite of all treatment, or even before there is time to carry any out. On the other hand, seemingly hopeless cases of this kind are sometimes pulled through by prompt attention. Patterson has recorded a case of mitral stenosis in which pulmonary embolism was simulated by the sudden onset of a superadded pneumonia, resulting in premature labour and death within twenty-four hours. Vinay describes another form of lung complication. The cases he records presented no complications except albuminuria, and the patients were suddenly seized with an exceedingly acute œdema of the upper portions of the lungs. In two cases death resulted in a few hours, while in a third premature labour came on and the patient recovered. He considers it to be of toxic origin, like eclampsia.

The results of kidney complications are both general and particular. Uræmic symptoms call direct attention to the kidneys, and suitable treatment is prescribed; but defective elimination does not always produce such marked results, and albuminuria frequently passes unnoticed, impairing the general nutrition and promoting degeneration of heart-muscle.

The diseases of the kidney arising from pregnancy, that have already been referred to, are more frequent with twins than in single pregnancies. This fact has been brought forward to show that a particular poison is excreted by the living fœtus.

Passive congestion of the liver has not, as far as I am aware, any direct influence on pregnancy beyond affecting the general nutrition, owing to its functional activity being impaired, especially if jaundice be present. It is conceivable, also, that the enlarged liver might exert a mechanical pressure on the heart, but I have not met with a case in which this has occurred. Premature labour may supervene in these severe cases, or the patient may go her full time, and only be saved from dying undelivered by the promptitude of the obstetrician.

The details of one of these cases have vividly impressed themselves on my mind. The patient at the end of the first stage of labour was pulseless, almost unconscious, and cyanosed to a greater degree than I have ever seen before or since. Delivery was only just effected in time, although the forceps were used before the os was fully dilated. Profuse hæmorrhage during the removal of the placenta relieved the patient for the time

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being, but she gradually sank, and died from asthenia on the tenth day.

Sudden failure, however, may occur without any of these complications being present. At the termination of the second stage of labour, the intra-thoracic pressure is sub-atmospheric, owing to the inspiratory effort that naturally follows the expulsion of the child. Suction power is thus exerted on the abdominal vessels, which are distended by the blood expelled from the contracted uterus, and the right side of the heart becomes dilated to such an extent that contraction is impossible, the patient dying suddenly from asystole before there is time to adopt any treatment whatever.

I have collected from various sources the records of twenty-six cases of mitral stenosis, in which the time of death has been given. Unfortunately in many instances the time is quite indefinite, the records merely stating that death occurred "shortly after delivery." Out of these twenty-six cases one death occurred during pregnancy, two during labour, and eleven in the first week of puerperium, four occurring in the first twenty-four hours after delivery.

Compensated cases of aortic insufficiency may go through several pregnancies without any signs of failure. I have one of these cases now under treatment. She has had five children, and everything appears to have been perfectly natural with the first four, but during the later months of the last pregnancy the heart showed signs of overwork, the patient complaining of palpitation, precordial pain, and dyspnœa. Absolute rest relieved the symptoms and enabled her to go her full time without failure taking place. Weakness of the heart-muscle, however, showed itself towards the end of the second stage and during the first two days after delivery, the patient having several slight syncopal attacks. As long as compensation is effective these patients pass through labour satisfactorily, and speedily rally when the third stage is over, even when the regurgitation is free and capillary pulsation present. But it is different when failure occurs in a previously compensated heart, because it probably means that the muscle of the left ventricle is degenerated, and the mitral orifice will sooner or later become dilated.

Pure aortic insufficiency is not a common disease in women of a child-bearing age, the mitral valves being generally involved as well. Under these circumstances the compensation is never so thorough as when the aortic valves are alone affected.

The symptoms when both orifices are involved are of a complex nature. Violent palpitation, cough, dyspnœa, occasional hæmoptysis, attacks of syncope, and precordial pain, amounting in some cases to angina pectoris gravior, may be complained of.

In three cases of combined aortic and mitral lesions that I have recently had under treatment the patients have complained of intense vomiting during the last few weeks of pregnancy. I have found it quite uncontrollable, and only relieved by parturition. In each instance the urine was scanty, but only a trace of albumen was present, and no headache was complained of. In one of them I was able to estimate the amount of urea, and found it nearly normal in quantity. Taking this in conjunction with the amount of albumen, I concluded that the vomiting was not the result of chronic or acute renal disease of pregnancy. It is surprising, however, to find how well some of these cases stand the strain when the heart-muscle is not degenerated.

The whole prognosis in cases of the aortic type depends on the condition of the left ventricle, for even if the lungs are involved, and their functions much impaired, lung symptoms take quite a secondary place as long as the hypertrophy of the left ventricle is sufficient. When it fails the case terminates rapidly; the patient may die undelivered, as in the cases recorded by Jaworski and Fellner, or, if she manages to struggle through labour, may die immediately afterwards—the termination of labour and the early days of puerperium requiring to be especially watched, as sudden death may occur, with or without any warning, from failure of the left ventricle.

Pure aortic stenosis is seldom met with in young adults, and it is especially rare to find the orifice much constricted at this age. When a case of this nature is complicated by pregnancy the special symptoms of the disease become exaggerated.

I have had under observation for the past four years a patient in whom these conditions are present. During this time she has had two children, the first having been born while she was in her teens. She suffered severely with both children, but nearly died with the second from

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epileptiform attacks during pregnancy and profuse *post-partum* hæmorrhage.

When the aortic orifice is less contracted regurgitation is present as well, and the symptoms partake more of the so-called aortic type. In all these cases, however, the patients are generally older, and some arterial degeneration is also present; therefore failing of the left ventricle more rapidly occurs, mitral insufficiency and lung complications resulting, as in ordinary aortic regurgitation.

Pregnancy in adherent pericardium is more dangerous to life than in valvular disease. As far as I am aware, I have only met with two of these cases. In one of them the aortic and mitral orifices were both diseased, and the pericardium was found on post-mortem examination to be generally adherent. It is therefore not a representative case. My diagnosis of the other case is only a clinical one, but I have watched the patient for over three years and am satisfied that it is correct. The patient was stated by her doctor to have had pericarditis seven months after the birth of her first child. When I first saw her, eight years afterwards, she was four months pregnant with her second child, and complained of constant fainting attacks, with palpitation and breathlessness. She was kept in bed for the remainder of her pregnancy, and went her full time. The symptoms increased in severity as pregnancy advanced, and for the last two months any movement brought on syncope; during the last month she was often unconscious for hours. The induction of premature labour was contemplated several times, but the patient was in too critical a condition. Labour pains set in sharply, and only lasted a very short time. No syncope occurred until after the child was born; she then suddenly collapsed, and very nearly died. She had subsequently repeated attacks of syncope, and was unable to sit up for thirteen weeks. She is still quite unable to take any exertion. Of course it is impossible to estimate the extent of the adhesions, or if the heart-muscle was involved, but there could not have been much fibrosis of the myocardium, or she would never have survived the ordeal.

Wilson records a fatal case in which the patient was pregnant for the third time, and had apparently showed no symptoms of failure, but she suddenly developed signs of syncope during the second stage, and died undelivered. The post-mortem examination showed a thickened generally adherent pericardium.

Fatty degeneration has been recorded as a cause of sudden death during labour and puerperium, but I have not met with any case in which this condition has been recognised before the patient became pregnant. In the cases recorded it was quite unsuspected, and death was instantaneous.

(*To be concluded.*)

WITH Dr. E. W. AINLEY WALKER AT GUY'S HOSPITAL.

General Pathology of Infection—III.

We considered last lecture some of the older theories of the nature of immunity, and discussed the earlier evidence bearing on this question. Thence we passed on to a consideration of the more modern views. Here we began by an examination of the Phagocytic theory, which was held by Metchnikoff, and of the evidence on which that theory rested.

We saw, however, that though invading micro-organisms may be taken up by phagocytes, they are not necessarily destroyed, but may survive and multiply within them. The phagocytes may thus themselves be killed, and the bacteria again set free into the blood and tissues to continue their invasion. It follows that the phagocytic process does not of itself ensure the destruction of bacteria.

Moreover, as I told you, it was shown that cell-free blood possesses a bacteriolytic action. Accordingly the phagocytic theory of immunity, on which the destruction of bacteria was entirely due to their ingestion by the phagocytes, became untenable.

The observations which threw doubt upon the view of Metchnikoff led Grohmann to the enunciation of the so-called Humoral theory. This held that the destruction of bacteria is dependent on the action of certain substances existing in the body-fluids, and not upon the phagocytic process.

The question was now taken up by Buchner and his pupils, who confirmed the observation that the body-fluids may exert bacteriolytic action both in the living animal and *in vitro*. Moreover, Buchner showed that blood which had been frozen and thawed, and the leucocytes thus broken up, was almost as actively destructive of bacteria as normal blood, although, of course, no phagocytic action could now occur. On the other hand, its power was rapidly diminished if it were slowly heated, and was entirely lost at 55° C. Accordingly he was led to attribute the protective action to a purely chemical action of certain substances which he termed Alexines, and regarded as being of the nature of active ferments.

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Buchner found these alexines to be extremely labile substances; to be, as I have said, exceedingly susceptible to the action of heat; to lose their power if substances favourable to the nutrition of bacteria were added to the solutions, or if the sera of different animals were mixed together; to require for their action the presence in solution of certain salts; and to be preformed in the serum, from which they disappeared on the precipitation of its proteids with alcohol.

Many of the latter experiments, however, gave contradictory or indefinite results, while Metchnikoff and his followers were continually producing further evidence to support the cellular theory. Thus they found that anthrax spores, freed from all anthrax toxins and inoculated in the rabbit, are rapidly destroyed by phagocytic action, and the animal remains unharmed. But if the spores be introduced, enclosed in a collodion capsule by the method of Sanarelli, and thus protected from direct attack by phagocytic cells, they develop to bacilli, which produce their usual toxins, and presently bring about the death of the animal concerned.

Yet many facts remained incapable of satisfactory elucidation on the phagocytic theory, while, on the other hand, the results obtained by its supporters, as much as those advanced by its opponents, rendered the purely humoral theory of immunity quite insufficient. Hence arose the view, attributable more especially to the influence of Bouchard, and which is usually spoken of as the humoro-cellular or cellulo-humoral theory. This theory forms to-day the only really tenable position on the general question.

The process involved consisted on the view of Bouchard in the simultaneous action of two forces, the bactericidal power of serum and the phagocytic activity of the leucocytes. Before the latter force could come into play there must occur in the acquisition of immunity a chemical alteration or modification of the blood-plasma. Certain bacterial products were supposed to produce this alteration in the composition of the body-fluids by gradual changes, which they brought about in cell-nutrition. The altered fluids then reacted on the bacteria in such a way as to destroy their power of resisting the attack of the phagocytic cells.

Metchnikoff had himself, in 1889, been driven to the admission that at least a part of the bactericidal action *in vitro* is attributable to substances present in the serum, but those he claimed were yielded to it by the leucocytes destroyed in the process of coagulation of the blood, and separation from it of the serum. On this question a whole series of monographs have appeared, commencing with the observations of Hankin and of Kanthack and Hardy in 1892, and carried on by Loewy and Richter, von Foder, Denys and Lecelf, Havet, Buchner, Bordet, and many others. Into these it is impossible to enter fully, but the results may be summarised as follows:

The fresh blood or serum of an immune, or an immunised animal, possesses a bacteriolytic action, and this action is greatest when the animal concerned presents a condition of marked leucocytosis, and is diminished when the leucocytic count falls below normal. Again, if sterile pleural exudates rich in white corpuscles be artificially produced—for example, by the injection of plant globulin into the pleural cavity of a rabbit—they are found to be possessed of high bacteriolytic power, even for bacteria on which the blood of the same animal exerts only a small, if any, lytic action. And it was shown that the bacteriolytic action of these exudates is associated with the presence in them of enormous numbers of leucocytes, for if these corpuscles be rapidly separated from the fluid by filtration the bacteriolytic action disappears, but is at once restored on their replacement. If, on the other hand, the leucocytes are not removed, but merely broken up and disintegrated in the fluid by the process of freezing and then thawing it again, bacteriolytic power remains.

Accordingly, we have the following conclusion—that leucocytic fluids possess bacteriolytic power outside the animal body; and cell-free fluids may exhibit similar action, but only actually appear to do so when they presumably contain the products of degenerated leucocytes. Bacteriolytic power is therefore evidently in close association with the reaction of the leucocytes.

In the passive immunity induced by the injection of immune serum Metchnikoff also claims a considerable share for phagocytosis, holding that the action of the serum consists to a large extent in a stimulation of the leucocytes and a consequent increase of their activity and numbers, such as we have seen can be induced in a less special manner by the injection of a great variety of indifferent

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substances. Pfeiffer, however, who found that anticholera serum possessed no antimicrobic power *in vitro*, but became rapidly bactericidal after injection in the living animal, showed that in the intra-peritoneal experiment the injected cholera vibriones are not infrequently almost entirely destroyed before the appearance of any considerable number of leucocytes can be determined. Hence he arrived at the conclusion that the serum owed its activity to the agency of other cells than leucocytes, and probably to the endothelial cells lining the peritoneal membrane. But in this connection it must be remembered that the endothelial cells also belong to Metchnikoff's group of phagocytes, being classified by him as of the class of the "fixed" phagocytes.

In intimate relation with the questions of bacteriolysis and immunity to bacterial infection are the phenomena of immunity to toxins.

At the lowest level in the scale of existence we already find certain forms of life that are unharmed by toxic substances which prove markedly and rapidly fatal to other forms. And here again we can by appropriate methods induce immunity in the susceptible forms. The bacteria themselves afford excellent examples of this principle. Thus the bacillus of fowl-cholera is destroyed by a solution of corrosive sublimate in a dilution of 1 to 25,000, while Friedlander's pneumo-bacillus is naturally immune to this solution, and is only killed when the dilution is reduced to 1 to 15,000. Again, Trambusti found that the latter bacillus could be accustomed—that is, immunised—to the presence of sublimate to such an extent that it could still exist in the presence of a concentration of 1 to 2000. In the same way Effront was able to immunise yeasts, and Haffkine to adapt the infusorian *Chilomonas paramœcium* to exist in strongly alkaline solutions. And many other investigations have given similar results.

An interesting example of the essential difference between this toxin immunity and bacterial immunity is found in the observations of Metchnikoff on certain larvæ which are immune to cholera toxin. Such larvæ, he found, support a dose of toxin which proves rapidly fatal to the frog. On the other hand, if living cholera culture be injected the larvæ die of general infection with doses from which frogs invariably recover. In the latter case the leucocytes of the frog attack and render harmless the bacteria, while those of the larvæ fail to achieve a similar success.

After the remarkable discovery of Behring, already mentioned, of the antitoxic properties of immune serum, an explanation of these facts was naturally sought in the occurrence of a pre-existing antitoxin in the blood of the animals in question. But numerous investigations absolutely failed to show the existence of such bodies in the naturally immune, so that Behring was able in 1896 to formulate the statement that the blood of naturally immune individuals possesses no antitoxic properties.

The accurate investigation of toxin immunity dates from the discovery of Roux and Yersin in 1888 of true bacterial toxins. Their observations, and others to which I have already briefly referred in speaking of the bacterial products, have helped to throw much light on the difficult question of bacterial antitoxins. It has been further shown that antitoxins similar in their properties, in their specific character, and in their general action to the bacterial antitoxins, can be obtained by the inoculation of toxins of non-bacterial origin. Such were the antitoxins obtained by Ehrlich against the powerful toxic bodies ricin and abrin.

As regards the mode of action of such antitoxins, the view originally put forward by Behring held that the process at work consisted in a simple neutralisation of the toxin, and was a purely chemical phenomenon. This position was supported by Roux and Vaillard in a series of experiments on the antitoxin of tetanus. Buchner, however, came to the conclusion that the antitoxin in some way inhibits the toxin in the organism without actually causing its destruction; for he found that mixtures of the two substances can be prepared which are harmless for mice, but prove fatal to the more highly susceptible guinea-pig. Behring, on the other hand, urged that in the mixtures of Buchner a true neutralisation of the toxin occurred; but that with the proportions taken there remained over a certain amount of the toxin which had not been neutralised, and which, though insufficient to injure mice, was still enough to cause the death of guinea-pigs.

The principle involved in this interpretation is one of the greatest importance to a clear conception of the events concerned in the causation of protective processes, namely, that whatever be the

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infective agent, or the toxin, and whatever the animal employed, there must always exist a certain minimal dose, quantities less than which fail to produce the usual infective or intoxicating action; that is to say, that in all cases the *basis* of immunity is already present in the animal. Moreover, Ehrlich, Cobbett, and others have definitely proved that the reaction of toxin and antitoxin—the neutralisation of the former by the latter—can take place just as well in test-tubes as in a living animal, and therefore necessarily must be a purely chemical event.

We have in this relation to keep most carefully and constantly in mind the clear and well-established difference which exists between antibacterial and antitoxic action in endeavouring to explain the process of protection. This distinction is most clearly marked, and all the evidence goes to show that the protective processes concerned are essentially divergent.

One and the same bacterium may be at one time a harmless saprophyte, say in the human pharynx, while at another it may gain local access to the tissues, and, producing there its toxins, give rise to an intoxication; or, again, it may, under suitable conditions, be induced to cause a general infection of the animal. But any given bacterium must at a given time fall under one or other of these groups as regards an individual animal. And the condition of the serum after infection is found to exhibit corresponding properties. Thus, if the bacterium falls into the second class and can only gain a local hold upon the tissues, it there produces toxins which diffuse throughout the organism and give rise to the formation of an antitoxin. On the other hand, if it acquire invading power superior to the natural resistance of the phagocytes, and cause a general infection, there will appear, in animals which tend to make recovery, a new induced reaction directed to the micro-organisms themselves. The serum then is found to have acquired anti-microbic action in addition.

Of these two properties—antitoxic and antibacterial—we shall consider first the antitoxic action.

In speaking of the protective substances of immune sera and of the views of Buchner, I used his term "alexines" to denote these substances. This, as perhaps appearing to involve the acceptance of a definite but ill-supported theory, we shall now relinquish in favour of the inelegant but more general term of antibodies, or anti-substances, retaining the name "antitoxins" to connote the antibodies of toxic substances. These antibodies have been in recent years most carefully investigated, more especially by the school of Ehrlich—himself the most brilliant investigator of the day.

The true bacterial toxins being in themselves difficult to prepare, and from their instability and complicated nature still more difficult to investigate, much of the evidence with regard to antitoxic sera has been obtained from the results of observations on other and more accessible substances which give rise to a similar formation of antibodies. Among these I may mention more particularly certain vegetable poisons, such as ricin and abrin, the venom of snakes and scorpions, and certain enzymes, notably rennet ferment. The evidence obtained from these and similar sources we shall now examine.

If a bacterial toxin be injected in successive and increasing doses it gives rise to the formation of an antitoxin by the animal. The action of the antitoxin so produced is found to be specific,—that is to say, it acts in general only against its own specific toxin. This *qualitative* action led at first and naturally to the view which was proposed by Buchner, that antitoxin was in some way derived from the injected toxin. And certain observations of Klemperer and others seemed to support the theory. The position was, however, shown by Ehrlich to be quite untenable on the following grounds:—Firstly, that we can, by the introduction of a given dose of toxin, obtain a quantity of antitoxin able to neutralise the original amount of toxin a hundred thousand times and more. And secondly, that the active toxin immunity induced will last for a considerable period, in some cases for years, or even for life, while passive immunity, which follows the injection of an antitoxin, is quickly lost; whereas if antitoxin consisted merely of a modified toxin, its length of stay within the animal body could not be affected by its manner whether of production or of introduction, but should depend solely on its amount.

Moreover, Roux and Vaillard showed that an actively immunised animal, whose blood had acquired a constant antitoxin content, rapidly regained this antitoxin level after repeated and

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copious venesections, even after the last remaining traces of the toxin originally injected must long have been abstracted from the blood in consequence of the frequent and free bleedings.

That the formation of antitoxin is essentially a *cellular* reaction is further proved by the observation of Salomonsen and Madsen that the antitoxin content of the blood could be increased by the administration of substances such as pilocarpin, which are known to stimulate the secretory activity of the body-cells, but could not possibly affect the injected toxins.

As the result of observations such as I have mentioned, Ehrlich was led to the opinion that these substances cannot be other than ordinary normal products of the cellular metabolism. Their production by the animal can be explained upon the following supposition, which forms the one and only necessary postulate of this theory—namely, that certain cells susceptible to the action of a given toxin contain particular elements or atom-groups, more or less independent and detachable, which possess a strong affinity for that toxin.

Now so long ago as 1885 Ehrlich had come to the conclusion, in considering the oxygen requirement of the organism, that the protoplasm of every living cell must of necessity contain numerous groups of such elements, destined in normal metabolism to attract and store nutritive substances which, as we know, circulate in the blood in very weak solution. To these hypothetical groups of elements he gave the name of lateral chains or side chains. Such chains could evidently also take up from the blood any toxic body for which they happened to have a strong affinity. Thus nerve-cells whose side chains possess a very strong affinity for tetanus toxin, abstract it from the circulating blood and incorporate it by their hypothetical side chains.

Now, toxin thus fixed maintains its attachment to the cell concerned for a considerable period of time. There must therefore be in the side chain a group of atoms in which one atom-complex—spoken of as the haptophore group—corresponds to the toxin and fixes it in much the same way as a key fits its lock.

But once this process of fixation has occurred, and the haptophore group is anchored to the toxin, the lateral chain is no longer free to exercise its usual function of attaching molecules of nutritive material; it becomes, so to speak, cut off from the physiological functions of the cell in question. The cell, therefore, to regain its normal functions, must constantly produce new chains. Now for such regenerative processes there is a general law, which Weigert formulated, according to which there occurs not compensation merely, but always *over-compensation* for what has been lost; so that more side chains are produced than have been fixed by toxin.

If, now, the new chains thus formed are constantly charged with fresh supplies of toxin by repeated and increasing fresh injections, a constant locking up and loss of side chains will go on which is continually compensated for and over-compensated, and the cells become thus "trained" to the production of a great excess of these particular elements. Finally, so many chains are formed as actually to become prejudicial to the metabolism of the cell, and this accordingly gets rid of them into the blood like any other waste or prejudicial product.

The chains set free into the blood compose and are the antitoxin, which by its strong affinity fixes the toxin then or subsequently present in the circulating fluids, and thus prevents its reaching organs which it otherwise attacks and injures.

This is the theory of Ehrlich. It is hypothesis and not established fact; but it provides us with a view to work from which is eminently logical and satisfactory. It rests on a recognition of the fact that processes akin and quite analogous to the formation of an antitoxin go on continually in the living body, and form a necessary part of normal physiology; and that the majority of intermediary metabolic products provoke a formation of new lateral chains in certain groups of cells, either in moderate amount, or, it may be, in quantity sufficient to give rise to an excess which is discharged into the circulating fluids as waste matter. Thus the blood-plasma must normally contain numerous substances which have resulted from the elimination of side chains by various tissue-cells. Analogous reactions can undoubtedly be artificially induced in the laboratory by means of various non-poisonous substances, and even with ordinary nutritive materials.

This view exhibits the production of immunity to toxin in the animal body as a process holding a position exactly parallel to that presented by the

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habituation of a bacterium or a protozoon or plasmodium to nutritive media containing substances normally prejudicial to their life and growth; and as depending on a gradual modification of the intimate physico-chemical processes of its metabolism.

The great beauty of this hypothesis lies in the fact that it at once explains (as Ehrlich himself has claimed) all the apparently marvellous and unique characters of the antitoxins. Thus their specific action, though they are not derived from the toxins, results from the fact that the condition and cause of the toxic action of the toxin lies in the special affinity of its haptophore for the haptophores of certain lateral chains of the cells which it attacks, for these are the lateral chains which constitute the antitoxin. Similarly we find here an explanation of the relatively great production of antitoxin which results from a methodical immunisation, and of the fact that the later injections only serve to increase the formation of antitoxin which is going on, as well as of the fact that the antitoxin content of the blood regains its former level after most copious and repeated bleedings. The theory also gives a reasonable explanation of the essential differences already noted between the active and the merely passive acquisition of immunity.

That toxin actually does become anchored to the cells in the way which Ehrlich has supposed was shown by Wassermann, who found that tetanus toxin mixed up and triturated with normal brain substance loses its toxicity by becoming "fixed." The lateral chains are adapted to the selection and fixation of nutritive substances, and toxins which excite the formation of antitoxins are, strange as it may appear, absorbed by the cell as though they consisted of simple nutritive material. Their toxicity is not due to the same atom-group as that which is attracted by the lateral chain and which is called the haptophore, but to an entirely different atom-complex of the toxin molecule, the so-called toxophore. For Ehrlich showed that toxins whose toxophores have been destroyed by artificial means, and which accordingly no longer have a toxic action, can still give rise to the formation of specific antitoxins, and this has been confirmed by Dr. Ritchie.

From this it follows that for the production of a given antitoxin the only elements essential are the proper haptophores, injected in sufficient quantities. Hence any and every substance, whether toxic or indifferent, which possesses the same haptophore will, on injection into animals, give rise to the formation of the same antitoxic substance. And this in turn will be protective not alone against the substance used for its production, but against every toxin which may happen to possess the same combining haptophore. This is the explanation of the reason why, though antitoxins are specific in their action, yet cases do occur in which an antitoxin may be found to have protective action for quite other toxins than that which actually gave rise to its formation (tetanus antitoxin against snake-venom, etc.).

The purely chemical nature of the reaction and the quantitative relation existing between a given toxin and its antitoxin was definitely proved by Ehrlich, who has shown, for instance, that the agglutinating action of ricin on the red corpuscles of blood is capable of quantitative neutralisation by its proper antitoxin. Similar facts have been established by a number of observers for various other antitoxic substances. The most decisive of these observations were those made by Morgenroth on anti-rennin and the rennet ferment, where, in the absence of all protoplasm whatever, the purely chemical nature of the action of the antibody is beyond question.

That the formation of such anti-substances is simply due, as Ehrlich holds, to an accentuation and increase of normal metabolic processes, is shown by the discovery of Röder and Hammarsten that normal horse's serum frequently contains a powerful anti-rennin formed under the action of the rennet ferment which has been absorbed from their own stomachs in the process of digestion.

The action of an antitoxin is to neutralise or chemically saturate its toxin; it does not destroy the toxin. For a toxin which has been made inactive by the addition of its proper antitoxin can be restored to its full potency by an exposure of the mixture to a temperature sufficient to destroy the antitoxic molecule. This usually occurs at about 68° C.

The lateral chains of cells, which have the power of taking up such substances as are provided with a proper haptophore, are now spoken of by Ehrlich as the cell-receptors. And since the normal products of metabolism, which are thus taken up

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for the nutrition of cells, are very numerous, the number and variety of these receptors must be very great indeed. But every substance which can find a suitable receptor in the living body will give rise to the production of its corresponding antibody if suitably injected in sufficient quantities to cause excess formation and the splitting off of new receptors from the cells affected. Accordingly a very great variety of different antibodies may be formed by the same animal. A good example of this is to be seen in the results which follow the repeated injection of bacterial cultures. Such a culture contains, besides the bacteria themselves, a number of different products of bacterial activity, and each of these which finds within the living animal a suitable receptor gives rise to the formation of its corresponding antibody. Thus, while the injection of a single chemical substance or a pure toxin results in the production of a univalent serum in the animal injected—that is, a serum containing a single antibody only,—the injection of mixed fluids like bacterial cultures leads to a multivalent action of the serum, which now contains a number of anti-substances in correspondence with the substances injected. Such are the antitoxins, antiferments, agglutinins, coagulins, and the like, which may be co-existent in a given serum.

Moreover if we inject into an animal a fluid containing cells or bacteria, these must be broken up and dissolved in the body fluids before the *intra-cellular* substances which they contain can come to act upon the body tissues and provoke the formation of the corresponding anti-substances. And if the cells be injected in a living condition, they must first be killed or die before these processes can be initiated; the antibodies then formed will be the antagonists *not* of the living cell, but of their dead materials. Accordingly, though they prevent and neutralise the harmful action of bacterial products or of cellular derivatives, they do not kill the bacteria or the cells injected; indeed, the immune sera, as they are usually called, frequently form admirable culture media for their own bacteria, although in other cases they appear to have some inhibitory influence on bacterial growth.

It follows, therefore, that the events I have described in speaking of the production of antibodies, while they give rise to the formation of substances protective for the organism concerned against the action of extra- and intra-cellular bacterial products, do not in any way lessen bacterial virulence or kill the bacteria themselves. Accordingly these events alone could never serve to put an end to the invasion of bacteria—which, as was shown by Pasteur, undergo a progressive increase of their virulence by a successful residence within the body—were they not assisted, and, indeed, curtailed by the intervention of another and more complex process, which we shall next consider. This is the process of bacteriolysis; that is, the destruction and solution of the micro-organisms themselves, which will form the subject of our next discussion.

Sudden Death from Enlarged Thymus.—Grawitz was the first to direct attention to the medico-legal importance of the enlarged thymus as a cause of sudden death. A communication from the pathologic institute of which he has charge describes two new cases. One was an infant who lived only two hours after version and birth, and the other was an apparently healthy six-month-old babe. Both died suddenly, and the only plausible cause of death was a very large thymus gland. In the second case the gland was in two lobes respectively 10 and 8·5 cm. long. The cases confirm the assumption that a large thymus may compress the trachea until it is actually flattened. If the head is thrown back, or the throat assumes any unusual position, the trachea becomes completely occluded and suffocation ensues, although the compression heretofore may not have caused any appreciable symptoms. This explains certain sudden deaths in the water, as the head is thrown unusually far back in swimming. This may also cause unusual pressure on the veins of the neck, which in turn is liable to cause congestion of the gland and consequent increase of pressure. Siegel has reported the case of a child who had suffered from dyspnœa for several weeks, finally relieved by tracheotomy and the introduction of a long tube extending to the bifurcation of the bronchi. The long cannula caused ulceration, and on the assumption that the enlarged thymus might be the cause of the dyspnœa, the gland was drawn up after opening the mediastinum, and it was sutured to the fascia over the sternum. The child was completely freed from all his troubles.—*Journ. A.M.A.*, December 13th, 1902.

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THREE LECTURES
ON
COMPLICATED FRACTURES AND THEIR TREATMENT.

Delivered at the Medical Graduates' College and Polyclinic, March, 1902.

By C. B. KEETLEY, F.R.C.S.,

Senior Surgeon to the West London Hospital.

LECTURE I.

COMPOUND FRACTURES.

The presence of an open wound communicating with the fracture.—The history of antiseptic surgery is, from its birth, closely associated with the treatment of this condition. It was especially on compound fractures that Lister made some of his first experiments in the wards of Glasgow Royal Infirmary, and the Germans first put his theories to the test, and, finding that the results were as he stated, played a strong part in converting the French, the Americans, the Italians, and the British themselves. The last were, as you may remember, somewhat slow to realise that the greatest improvement surgery had seen in either historic or prehistoric times had just had its nativity in their own island, in their own days, fathered by their own countryman. In the late seventies, while some of the famous teachers of London, Edinburgh, and the provinces, who ought to have been singing the song of Simeon, were indulging in cheap sarcasm about germs, Nüssbaum, of Munich, and others were telling how the adoption of Listerism had converted compound fractures from among the most deadly of calamities into the most benign.

Now to business. A compound fracture having occurred, the first indications are—

To check hæmorrhage,
To purify,
To adjust and (if necessary) fix internally,
To dress,
To fix externally,

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and, at the same time, not to lose sight of the general condition of the patient, or to overlook injuries in other parts of the body.

Hæmorrhage.—This has often ceased before the surgeon sees the patient. But even then, if the latter has been left blanched and pulseless, or with a very frequent and running pulse, steps should be taken to (1) prevent the recurrence of bleeding, and (2) to refill the blood-vessels. The latter process can often be carried on by assistants simultaneously with the surgeon's personal attention to the local injury. In connection with this subject I would recommend you to read Mr. Beddard's paper on "Transfusion," published in 'Guy's Hospital Reports' about two years ago. It will bring you up to date in a subject important to medical men in every variety of practice, and you will find it most interesting.

A pint of warm neutral saline can generally be persuaded to enter and stay in the adult rectum, which, unless the patient's condition is too depressed at the moment, can sometimes be advantageously washed out first, through a speculum. The same fluid can also be injected into the subcutaneous tissue of the axilla, the groin, the flank, or the abdomen, or into one locality after another, if the operation has to be repeated at intervals of a few hours, as it often should be in very severe cases. For intra-venous transfusion I prefer the internal saphenous to the median basilic or cephalic. The internal saphenous is larger, and is very easy to find owing to the practice one gets in doing Trendelenburg's operation for varicose veins. I do not think there is much need for strychnia in these cases. Alcohol I regard as useless, and sometimes worse; but that is a wide subject.

Anæsthesia is often essential. But it should be used economically. At least the preliminary cleansing can often be done without it. With proper assistance it need only be profound during the setting and fixing. It is remarkably rare for any large named artery or vein to require ligature in connection with a compound fracture, unless amputation is done. If, however, the necessity should arise, any incision required should be sufficiently free for its purpose, it should generally be longitudinal, and it may be utilised in cleansing and even in adjusting and in wiring or joining the fragments.

Clamp forceps may be placed on small vessels and points. On their removal the bleeding has generally ceased. If the hæmorrhage is deep and inconveniences the operator, the limb should be elevated and an Esmarch's tourniquet applied and left in place till the dressings and compression bandages are on. But the surgeon should not then go home and forget it till suddenly in the night a horrid fear and remembrance seizes him. This fear, in the experience of myself and my friends, has always proved a mistaken one—happily. But to prevent both the fear and the accident, I make it a rule that Esmarch's tourniquet should not be hidden beneath either clothing or mackintoshes.

Sometimes mere elevation during operation is better than using a tourniquet. In operating on the foot in a case in which compression after operation would be undesirable I sometimes place the patient on the floor, and the foot on a table. The larger vessels then bleed enough to betray their whereabouts, and not enough to harm the patient or inconvenience the surgeon.

The hot lotions used in cleansing tend to stop oozing from very small vessels.*

Temporary gauze tampons are also very useful. Each should be marked by a catch forceps. If one is left behind and forgotten, suppuration is inevitable. Of course, to leave them behind on purpose and remove them at the proper time is quite a different thing.

Purification.—This often means something more in the case of a compound fracture than in the case of an ordinary operation wound: for the former may be full of dirt, and contain tissues so bruised and otherwise injured that the question of whether they will live or not may arise, and consequently that of whether they should be left or removed.

Regard should be had to the position of the fracture. A recent wound in a clean, healthy person, and entirely covered by clean linen, will remain usually for some time practically an aseptic wound. Conversely a smashed head, or ungloved hand, or an ankle, polluted by street dirt, is generally infected at least by micrococci of suppuration if not by the tetanus bacillus.

In the former class of case, cleansing, though it should always be careful, is often very simple. A little gentle rubbing with 1 in 20 carbolic washed

* Ref. to 'Practitioner,' Feb., 1879, p. 113.

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off with 1 in 2000 sublimate solution, the latter used warm and for a few minutes, suffices.

But in the dirtier class of cases considerable time and great pains must be employed, and even operative measures may be required for the mere purpose of cleansing.

1. The skin for a considerable area round the wound should be cleaned with benzine, or with turpentine, on lint or cotton wool.

2. This should be wiped off and a thorough washing, for several minutes, effected with soap, and hot water softened to a feeling of slight oiliness with liquor potassæ, or with common washing soda. I have never seen any harm come of the soap-suds getting into the wound.

3. Swabs dipped in 1 in 20 carbolic should be used.

4. All the carbolic should be carefully washed away with swabs dipped in 1 in 2000 sublimate solution.

All the solutions should be warm, more especially if the area to be cleansed is large. It cannot be right to spend ten minutes in chilling a large surface of the body of a patient suffering from shock or hæmorrhage; nor is cold a good thing for the vitality of raw tissues.

A superficial wound can sometimes be cleansed itself at the same time as the skin around it, though the same swabs should not be used to both.

As a preparation for the cleansing of deeper wounds, the skin around, previously purified, should be protected by antiseptic or aseptic towels. No probe should be inserted until the superficial parts it will have to pass through are cleaned. The depths and extent of the injury having been approximately made out, the surgeon should ask himself the question, "Am I certain that no deep infection has taken place?" If the answer is "Yes," well and good; if "No," then he must proceed.

The custom was, and with many persons still is, to syringe out the deeper spaces with antiseptic lotions. This may be successful even in long unbroken series of cases, but it is sometimes unsuccessful. It may even be harmful by forcing the dirt in deeper and deeper, without, after all, disinfecting it. Swabbing is better. But to swab effectually the wound may have to be enlarged, and even fresh wounds made, *e. g.* on the opposite surface of a limb, or to deal with a cranial fracture. The position and direction of these wounds should, of course, be determined by anatomical considerations. In the limbs such incisions should generally be longitudinal. In the head their direction is much governed by the line of the fracture. The original scalp wound is sometimes too small and too inconveniently placed to be of any use at all except as an opening through which the position and direction of the line of fracture may be determined.

There are no compound fractures in which the bone itself so frequently requires cleansing as those of the skull. Dirt and hairs and germ-infected blood-clot are often trapped in the fissure. I never use a trephine for such cases, but shave off one edge of the crack with an ordinary small chisel and mallet.* This suffices to permit not only cleansing, but elevation of depressed fragments, as well as extraction of spiculæ or of loose pieces. When the scalp is not too much bruised, the patient a healthy subject, and the loose fragments aseptic, they should be replaced after elevation.

When I wish to preserve a loose fragment of bone for a time before replacing it, I sometimes, especially if the piece is large and the time is to be long, make a slit in an asepticised skin area, and slip the bone through into the subcutaneous tissue till it is completely hidden. For smaller pieces and for shorter periods, warm boracic lotion or "neutral saline" does very well, though they, of course, rapidly cool unless attended to.

It is not always necessary to shave hairy parts. The hair is more easily disinfected than the skin; nevertheless its removal is often required owing to its tendency to get in the way or into the wound.

Purification of the wound when the fracture is in a limb.—The general directions given above must be followed. Cotton-wool swabs leave pieces behind on the rough ends of the bone fragment. Such pieces should be carefully removed, and the final cleansing performed with gauze, and the douche or Higginson's syringe. Small pieces of gauze should be used for deep parts, and these may often be conveniently put on the ends of forceps or sponge-holders. In order to clean all round the bone ends it is sometimes necessary to turn them right out of the wound, but it is generally preferable to make a counter-incision. When there is any fear of suppuration, a fairly large incision on the

* 'Medical Press and Circular,' December 31st, 1890.

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posterior surface of the limb, opening right down to the fracture, is a wise precaution against bagging of pus. A large short drainage-tube should be placed in it.

Drainage-tubes should not be used unless there is special reason to fear suppuration, *e. g.* unless a long interval has taken place between the occurrence of a comminuted fracture and the process of disinfection. Half an inch diameter tubes are a reasonable size in the leg, especially for such as go through and through. Short tubes may be larger. Safety-pins should be put through each outer end, and kept off the skin by gauze. The judicious use of drainage makes it safer to leave behind fragments of bone which may, after all, not live. By injudicious use I should mean more especially the arrangement of the tubes in such a way that they keep the soft tissues more or less off the bone, which must adhere to these soft tissues if it is to live.

Never be in a hurry to use either the saw or the bone forceps in adjusting a compound fracture in one of the limbs. Large fragments, even though completely or nearly completely detached, frequently re-adhere and live. So also do long extruded ends, though stripped of periosteum, if carefully replaced. The result depends almost entirely on the degree of asepsis achieved. The rarity of necrosis after simple fractures, however severely comminuted, proves this.

In the course of cleansing the wound the opportunity should be taken of clearing out blood-clots. Not only do they interfere with adjustment, but should infection take place they lead the germs far into the intermuscular spaces, and thereby make the suppuration diffuse. Ogston long ago demonstrated the great distance to which effused blood will spread after a fracture.

Adjustment. — The X rays have shown how greatly we have deceived ourselves in the past as to the degree of accuracy with which our treatment has brought the fragments of bone together. A French author relates the case of a fractured leg, occurring in his own practice, in which he imagined the result to be practically perfect. The functional result was actually very good. So also was the appearance of the limb. But, with the aid of a skiagram, he discovered that the only union was between the upper fragment of the tibia and the lower fragment of the fibula. I show you here a radiograph in which the upper and lower fragments of the tibia appear to barely touch each other. Nevertheless the appearance of the limb and the functional result are as perfect as in the case related by our French *confrère* ('Centralblatt für Chirurg.,' about February or March, 1901). But I must warn you that, for some reason, optical or otherwise, the X rays exaggerate defects in the adjustment of fractured bones. They have been described as being "as truthful as a photograph." But what can be more deceptive, more practically untruthful, than many photographs?

When you are going to have a skiagram made of some fracture which you have treated, always warn the patient of this tendency to magnify defects, or, when the print comes home, you may find him horror-stricken, and perhaps even contemplating an action at law against you.

In adjusting a fracture which is compound we have this advantage, viz. the position of the fragments can always be palpated, and can often be seen. For palpation it is safer to use a boiled metal instrument than the finger. And, as adjustment of a compound fracture is not an operation requiring great delicacy of touch, the operator and his first assistant can wear boiled cotton gloves.

One fragment often projects through the wound, especially if the bone broken be the shaft of the tibia. After purification, reduction should be attempted by extension with counter-extension, and, if necessary, the surgeon may assist by pressure with his fingers or by pulling the lips of the wound out of the way with blunt hooks. If the knife has to be used a very limited use of it generally suffices. I have already spoken against a too speedy resort to the saw and bone forceps. The projecting bone having gone back, some steel instrument should again be employed as a probe to find out if the fragments are in good relative position without any of the soft tissues lying between them. If they are not right they must now be put right, and, if necessary, the wound enlarged for the purpose. The same rules for cutting the soft parts apply here as in cleansing. The ordinary instruments required in most operations will be needed, viz. retractors, blunt hooks, dissecting forceps, scissors, scalpels, needles, needle-holder, and hæmostatic forceps: and, in addition, necrosis forceps, bone forceps, and a small saw, or a chisel and a mallet. The cutting power of a chisel is obstructed by the

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"spring" of a broken bone. Further, you should have a bradawl, a narrow director to match it,* stout silver wire, and will you excuse me for also recommending my fracture pins? You may also provide yourself with some plated screws. Here are some with a removable handle I have had made to fit them.

Have (1) the common instruments; (2) the bone instruments; and (3) the hæmostatic forceps, in three separate trays, and get into the habit of not mixing them up during the operation. A capital needle-holder is Doyen's, which will hold a curved needle at any angle. A stout blunt hook with a large curve, *e. g.* of an inch and a half radius, is occasionally useful.

If all bones were solid throughout, like the os calcis, simple screws of plated steel might be used in nearly every fracture requiring internal fixation. But the shafts of the long bones are more or less hollow pipes, only strong in the shell. When, therefore, they are broken, even if there is no comminution, neither the screw nor the nail is always a perfect instrument for uniting them. This is a pity, because screws and nails are more easily put in than a wire suture, and do not need so large a wound. Indeed, they need in some cases no wound at all except a trivial puncture. Nevertheless, when the screw or the nail *will* fix fragments firmly in good position, it should be preferred, except in very superficial bones, such as the patella and the olecranon.

Where the bone lies deep, *e. g.* as in the case of the femur of a thick, fleshy thigh, suturing with wire is only easy when there is a large wound, unless a second wound is made on or towards the aspect of the thigh opposite the first wound. If a surgeon not used to suturing bones commences with a deeply placed fracture of the femur, he is apt to spend so much time and manipulation over the operation as to run the risk of wound infection before he has done.

Having regard to the above considerations, sometimes even when the fracture is compound, and a wound exists through which, with a little enlargement, there would be room to perform a suturing operation, and, with no enlargement at all, to insert screws,—sometimes even then, I say, I prefer to let the wound alone, and, after taking care that no soft part intervenes between the fragment, I use the L-shaped pins I originally devised for simple fractures. These, as I shall explain by-and-by, penetrate the bone at some distance above and below the fracture, so that each pierces and holds the two opposite sides of the bony cylinder, *i. e.* two layers of compact bone.

To return to the operation of wiring. I have never yet met with a healthy human bone which could not be perforated with a joiner's bradawl, sharp and of good quality, *e. g.* such as you would get at Buck's, and it can be disinfected by boiling as easily as the most modern surgical instrument with blade and handle all in one piece. I used to perforate the wooden handle with a fine drill, and tie blade and handle together with wire before boiling them. But I often found the ordinary bradawl blade too short. This is one reason why

I had constructed the patella instruments I now show you.

For hard bones a borer was made for and given to me by my friend Dr. Charles Firth, of Gravesend. It is an ordinary twist drill one eighth of an inch in diameter with a half-inch slant, to which has been added a brass-milled head about one inch in diameter. At the end of the drill a small hole is bored to carry the wire on withdrawing the drill. But if this hole is made large enough for a *thick* wire it dangerously weakens the drill. This drill should be used like a gimlet, not like a

* I recommend my own "patella case" (*see* 'Lancet,' December 10th, 1892). It contains a bradawl with an extra long blade, and a director a French-catheter size smaller.

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bradawl, and lightly and rapidly rather than forcibly.

I gave up Archimedean and other drills on account of the trouble their cleaning requires. Of course, a surgical engine, driven by electrical or other force, is the quickest and easiest way of boring bone, but it is seldom at hand.

When the bone has been perforated, the director is passed through it and the wire along the director. If several wire sutures have to be passed, it is generally advisable to get them all into place first before twisting up any. I give each suture three twists of 180° each,—just equal to one and a half times round a clock-face and always in the direction opposite to that taken by clock hands. In this way it is not necessary to make a special effort of memory every time a wire has to be removed. Not that in such a case it is usual to untwist the wire. It is more common to cut it. If the wire is to be left permanently, its ends should be tucked into the fissure, in which a little space may be cut for them. When the wire lies superficially, *e.g.* between the olecranon and the skin, it is almost always desirable to remove it ultimately. The removal should be postponed as long as possible, and effected in the following way:—(1) cut or untwist; (2) see-saw the wire to and fro to loosen it; (3) grip and twist one end round the blade of a pair of hæmostatic or of necrosis forceps; (4) pull out in a direction from the hole out of which the wire is emerging towards the hole into which it is disappearing. This lessens the risk of re-fracture.

Sutures well covered with muscle or fat seldom need meddling with. Concerning the removal of screws or pins nothing needs to be said, except that in cases when it would risk tearing the bone fragments apart it should be delayed.

To show how careful one should be about this point, I may say that when refracture has been necessary for mal-union, unless there has been no leverage, I have never had any difficulty in effecting it, apparently in the original place, within three months of the original accident.

I may here mention incidentally that operative treatment of fractures by adjusting the fragments precisely shows that the union of fractures is a shorter process than used to be supposed. A femur which had been set by operation was accidentally refractured without displacement. There was firm union in a fortnight although the patient was an adult. But a long period elapses between the time when a bone is united and the time when it is strong enough for the limb to be used. Part of this can be utilised for strengthening the muscles by rubbing and gentle passive exercises.

These observations are confirmed by experience of osteotomy.

When the processes of adjusting and wiring or pinning have been easy and short, the external wound may be closed at once without drainage, but not when the operation has been prolonged and difficult. It is then safer to defer suturing of either a part or the whole of the wound, and to lightly insert iodoform gauze rather as a drain than as a pack. After four days this gauze may be removed, and, if all looks well, the wound closed except at one corner which may be left open.

In deciding how and when to close the wound, regard must be had to the depth of the bone and to the condition of the wound. The deeper the bone, *i.e.* the thicker the layer of muscle over it, the less care one needs to take to close the wound. But great care should be taken to promptly cover such bones as the tibia, the patella, and the olecranon. Sometimes the soft parts are so injured as to necessitate a plastic operation. Sometimes the extent of the plastic operation needed cannot be estimated at first, because time is required to show the degree to which bruised and lacerated parts will recover. All prominent superficial bones require to be *well* covered. A mere Thiersch's graft is not always sufficient. It is better to loosen a flap or bridge of skin and fat and shift it over the exposed bone. The place from which this flap or bridge has come has usually a base of strong fascia lying on muscle. Thiersch's grafts do well enough to cover this. Such grafts are generally taken from the front of the thigh. There is thus, metaphorically, a process of robbing Peter to pay Paul, and plundering some other saint to repay Peter.

In all plastic operations the prime conditions for success are—(1) to avoid sepsis; (2) to avoid tension; (3) to obtain sufficient material.

Chloretone in Post-operative Vomiting.—During the past year Bickle has adopted the practice of giving fifteen grains of chloretone two hours before operating with satisfactory results.—*Therapeutic Gazette*, October 15th, 1902.

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A CLINICAL DEMONSTRATION

ON THE

TUBERCULAR AFFECTIONS OF THE SKIN.

Given at the Medical Graduates' College and Polyclinic.

By E. GRAHAM LITTLE, B.A., M.D., M.R.C.P.,

Physician for Diseases of the Skin to St. Mary's Hospital, and Lecturer on Dermatology in the Medical School; Senior Assistant Physician and Physician for Diseases of the Skin, East London Hospital for Children, Shadwell.

GENTLEMEN,—I have endeavoured to bring together this afternoon a series of cases illustrating the manifold appearances which tubercular affections may assume in the cases in which the skin is primarily or chiefly affected. My material is mainly drawn from the skin department under my charge at the East London Hospital for Children, and as these affections are more common in early than in adult life, they appear with exceptional frequency in such a department.

The tubercular diseases of the skin at present recognised may be grouped as follows:

a. Bacillary. In which tubercle bacilli have been demonstrated.

1. Lupus vulgaris.
2. Acute miliary tuberculosis of the skin (very rare).
3. Scrofuloderma. Tubercular gummata.
4. Tuberculosis verrucosa cutis.
5. Lichen scrofulosorum.
6. Bazin's disease.

b. Non-bacillary. In which tubercle have not yet been demonstrated, but in which a tubercular origin is probable. "Tuberculides."

1. Acne scrofulosorum vel scrofulosa.
2. Lupus erythematosus?
3. Dermatitis gangrænosa infantum?

a. BACILLARY.—*Lupus vulgaris* is the commonest type of tubercular affection of the skin, and the most indisputably tubercular. The old division into two classes of "lupus exedens" and "lupus non-exedens" is merely an expression of the clinical fact that some cases of lupus ulcerate and others do not, and is not worth retaining. The fact that lupus is tubercular in origin has been incontestably demonstrated by the following observations:—(1) Tubercle bacilli have been found repeatedly in sections of typical lupus. (2) Patches of lupus have reacted in a characteristic manner to injections of Koch's tuberculin. (3) Inoculations, accidental and experimental, with tuberculous material have resulted in the formation of lupus nodules. (4) Portions of lupus tissue, triturated into an emulsion with sterile broth, and injected into guinea-pigs, have produced in them generalised tuberculosis.

Lupus may appear on any part of the integument, but is commonest on the face, and most rare on the scalp, the genitals, and the palms and soles. It may attack mucous membranes, very rarely primarily, but not uncommonly as a secondary extension from adjacent lupus of the skin. It thus is often exceedingly difficult to distinguish these cases from syphilis, and I have brought up a case from Dr. Pringle's clinique, which was diagnosed in the first instance as a case of syphilis, a diagnosis which was modified only after the complete failure of antisyphilitic treatment. I showed a similar case * at the Dermatological Society of London in which the opinions of members present were almost equally divided as to whether it was lupus or syphilis, and here also the decision was made only when a rapid improvement with mercurial injections resulted. A distinction to be relied upon to discriminate in such doubtful cases is the greater chronicity of lupus; the rate of progress of this as compared with syphilis being, in Payne's graphic simile, "as the hour hand to the minute hand of a watch." Another distinction in cases affecting the mucous membrane of the mouth, especially the hard palate, to be remembered is that lupus never corrodes bone, while syphilis frequently does this; and, finally, the reaction to antisyphilitic treatment is sometimes an ultimate means of diagnosis, as exemplified above. The origin of the tubercular invasion in lupus is probably twofold,—from without by inoculation of the skin with tuberculous matter, and from within by derivation from visceral or bone tuberculosis. I have seen two interesting examples of the first category lately. In one, a woman developed a lupous nodule (sections of which showed typical lupous architecture) on the side of the nose, the seat of an abrasion by ill-fitting pince-nez

* 'British Journal of Dermatology,' vol. xiv, p. 24.

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spectacles. In the second case* an infant had apparently become infected in the hand by crawling about on the floor of a room in which a tuberculous patient had lived, and a short time previously died. In the second category I would place the two cases which I have here.

1. Rose W—, æt. 10. She has now multiple patches of lupus vulgaris on the arms, back and abdomen, and legs. Quite recently the upper lip and interior of the nostrils have been attacked. She was kicked on the leg when five years old. This did not heal, and eventually she had tubercular disease of the tibia, which was scraped for this reason. During the last four years patches of lupus have appeared on the sites in which they are now present. She has enlarged glands in the neck and groin. There is no family history of tuberculosis.

2. Irene G—, æt. 7. She has scattered patches of lupus vulgaris on the thighs, buttocks, and arms. There is evidence of old-standing disease of the bone in the right leg. This was treated surgically. The patches of lupus appeared afterwards, and fresh places are still developing. She lives with an aunt who has consumption.

The treatment of lupus vulgaris has undergone important modifications within the last three years, since the application of light rays has been discovered. Both the X rays and the Finsen light have been used in this way. The results obtained with the Finsen light have been better cosmetically than those achieved by any other means. This was certainly the verdict of all who were in a position to make a comparison between this treatment and others in a number of cases demonstrated at the Dermatological Congress in Paris in 1900. It is the treatment of election in lupus of the face, where unsightly scars are a serious misfortune. The X rays are probably nearly as useful, and in certain cases, where mucous membranes are involved, are preferable, as providing deeper penetrating powers. The X rays are also especially indicated in cases where there is much ulceration. Both forms of light treatment do not succeed well in cases where there is much scar tissue, often the result of previous scarification or scraping. Where the nodules are very small early excision is probably the best method, even on the face. In rapidly advancing lupus with much ulceration feeding with thyroid extract sometimes effects remarkable cures, especially in children. I have here a child who has a tubercular ulcer of this kind covering the greater part of the right leg. This is skinning over rapidly under thyroid treatment. Obviously also measures to improve the general health are important adjuncts to the local treatment; and many cases, especially of ulcerative forms, should have a preliminary six weeks at Margate before commencing any treatment.

Acute Miliary Tuberculosis of the Skin.—This is an extremely rare disease. A case reported by Leichtenstein is quoted by Fox ('Brit. Journ. of Derm.,' vol. xii, p. 396). It consists of an "eruption of acneiform papules, containing tubercle bacilli, evolved in the course of acute miliary tuberculosis." A somewhat similar case was reported by Hall, of Sheffield ('British Med. Journal,' September 28th, 1901).

Scrofuloderma; Tubercular Gummata.—Scrofuloderma is a term somewhat loosely used to designate deep-seated small abscesses, which often break spontaneously, and in the vicinity of which tubercular skin lesions are a frequent later development. These secondary patches are found, for example, near the sinus left by operation for tubercular pleurisy, or about the anus in ischio-rectal fistula, or in the neighbourhood of discharging tuberculous glands. I have brought up a case of exceptional interest in which numerous tubercular gummata, or what I take to be such, are found in an infant with a synchronous eruption of acneiform lesions, like those of acne scrofulosorum. Tubercular gummata of this type have quite lately* been demonstrated by Drs. MacLeod and Ormsby to contain bacilli, and in the same paper in which this observation was made the lesions of a case of acne scrofulosorum were examined and compared with the findings in the first case. With the exception that bacilli were found in the first and not in the second the appearances are identical. This present case, in which both types of lesion occur simultaneously, taken with the observations quoted in the paper, may be said to add a strong argument for the correctness of the view that both are tubercular manifestations.

* 'British Journal of Dermatology,' vol. xiv, p. 265.

* 'British Journal of Dermatology,' vol. xiii, p. 367.

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Tuberculosis verrucosa cutis. Example: Post-mortem wart.—I have not been able to bring a case to illustrate this affection, but you will no doubt have seen cases yourselves in the dissecting and post-mortem rooms. Tubercle bacilli have been demonstrated in sections of these lesions.

Lichen scrofulosorum.—This is a very rare affection, and I am unable to bring a case of it. I, however, hand round a water-colour drawing which gives an excellent idea of the appearance of the disease, which has recently been demonstrated to be bacillary, and therefore has been removed from the second group to be included with those in which bacilli have been proved to exist. The disease is characterised by groups of very small follicular pink papules arranged in a circinate manner, found usually on the abdomen, but in children not infrequently on the limbs, the abdomen being free. When it occurs in this grouped manner the only disease with which it can be confounded is the corymbose syphilide, which is also a rare type. It is usually unattended by subjective symptoms, and tends to disappear as the patient's general health improves, to recur when the health is deteriorated. Thus I have seen it in two brothers * who had the eruption almost every succeeding winter; they were adversely affected by the cold, and had "chilblain circulation." Other tubercular affections are frequent concomitants of lichen scrofulosorum. Jadassohn, quoted by Fox,† found in nineteen typical cases of lichen scrofulosorum only one who was certainly free from tuberculosis. Fox has seen it in association with multiple tuberculous lesions and with acne scrofulosorum. Tubercle bacilli have been found in this type by Jacobi, Wolff, and Pellizari (quoted by Fox, loc. cit.). The case from which this picture was drawn occurred in a boy of ten.‡ He had no enlarged glands, and no history of phthisis in the family could be obtained. He was subject to bronchitis, however, and he had a possibly scrofulous ulcer which had persisted for two months on the cornea of one eye. Sections from the affected skin were examined histologically. There were numerous giant-cells seen, but no bacilli. Experimental inoculations were made in two guinea-pigs with tissue derived from this case. One died within a few days, from other causes probably than the injection; the second animal survived for six weeks, and was then used for another experiment.

Erythema induratum, or *Bazin's disease*, is another rare type of a disease which is in all likelihood tubercular. Bluish nodules and plaques are formed in the skin; the skin surrounding them is deeply infiltrated, and feels firm and tough to the touch. The most usual position for their formation is the calf of the leg in young and often anæmic girls. It is much more rare in males, but I have had a case in a boy.* Other symptoms of tuberculosis are commonly found. Thus in my case there were peritonitis and synovitis, both almost certainly tubercular. The indurated nodules may persist as such for an indefinite period, or they may break down into ulcers which are very intractable. Tubercle bacilli have not as yet been found in cases of this type, but their histology is typically tubercular, the patches react in a characteristic manner to injections of tuberculin (Jadassohn † and Mantegazza ‡), and portions of tissue from the disease have produced tuberculosis when injected into guinea-pigs (Fox § and Thibierge ||). Mantegazza therefore claims that this type should be classed with the scrofulodermata rather than with the tuberculides, to be described immediately. It may be considered for the present as the connecting link between the two classes of bacillary and non-bacillary tuberculous affections.

b. NON-BACILLARY. — The general name of "tuberculides" was given by Darier in 1896 to this class of cases—that is to say, "to the different eruptions associated with tuberculosis, but apparently non-bacillary." It was therefore an entirely provisional classification, and the term "tuberculides" connotes, according to Darier, "eruptions occurring only in tubercular persons, with an unknown pathogenesis," and contrasted by him with the "tuberculoses" of the skin in which the bacillary pathogenesis is demonstrated. It follows from this definition that types which have previously

* 'British Journal of Dermatology,' vol. xiv, p. 12.

† Ibid., vol. xii, p. 386.

‡ Ibid., vol. xii, p. 167.

* 'British Journal of Dermatology,' vol. xiv, p. 93.

† Quoted by Darier, Fourth Congress of Dermatology, 'Comptes Rendus,' p. 148.

‡ 'Annales de Derm. et de Syph.,' June, 1901, p. 498.

§ 'Brit. Journ. of Derm.,' vol. xii, p. 383.

|| Quoted by Fox, loc. cit.

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been included in this class must be transferred to the first group as soon as the quest for bacilli in cases of such types has been successful. It is for this reason that lichen scrofulosorum and Bazin's disease are here no longer described as tuberculides, though in most text-books they are still included in the doubtfully tubercular group to which this general name is given. Fox, who has studied this subject with especial keenness, has described* a type of disease seen not infrequently in children who usually show other signs of scrofula, and who develop an acneiform eruption to which he has given Bazin's name of Acne Scrofulosorum. The eruption is "indolent in its course, and disseminated for the most part sparsely and without grouping, affecting the limbs particularly, and especially their external aspects, the lower extremities mostly, and notably the buttocks and the regions immediately above. The lesions appear successively, or by a subacute outbreak; they are small papulo-pustules, inflammatory, and seated about a hair-follicle, and acuminate in the early stage. They mostly develop a slight crown of pus, which dries up and often shells out with a central follicular plug, leaving a crater. The papules in involution become flattened and more irregular in outline, simulating lichen planus, and they tend to leave stains or faint scars." I have taken much interest in these cases, and have seen a considerable number of them lately. I believe them to be comparatively common, at any rate in the East End, where poverty is the rule, and therefore tubercular diseases of all kinds abound. In the great majority of these cases other signs of tubercular disease are to be found. Fox states that out of twenty-four cases shown at the Dermatological Society of London, half were affected with "adenopathies or other marked tubercular lesions." My own experience would make the association with other tuberculous symptoms much more constant than these figures indicate. The treatment of these cases is often very satisfactory, the eruption disappearing under a course of cod-liver oil and iron, especially when combined with a visit to the sea-side.

Lupus erythematosus.—The relation of lupus erythematosus to tuberculous disease is still too disputable for profitable discussion here. Suffice it to say that many authorities consider that it has an undefined connection with tuberculosis.

Dermatitis gangrænosa infantum.—This is a rare disease, and I have not been able to bring an example to show you, but I pass round a water-colour drawing of a case, from which you will derive an accurate impression of the appearance of the lesions. You will see there depicted a number of small round ulcers, sharply punched out and deeply excavated. The floor of the ulcer is very red and smooth. The commonest antecedent of this disease is varicella, and it has also been called varicella gangrænosa. As it may, however, arise independently of varicella, it is better to use the name here given. The cases are certainly rare, but Payne* pointed out some years ago that almost all the cases which had died had succumbed to general tuberculosis. The *Bacillus pyocyaneus* has been found in some cases, but I am not aware that tubercle bacilli have been noted in the ulcers themselves. I have had two instances of this affection. The first case,† the drawing of which you have seen, was in a girl aged nine months. There had not been antecedent varicella, and the child had been well until the week preceding the eruption, which had commenced with a pustule on the dorsum of the foot. This broke very soon, and a large, deep hole was left in its place. Similar lesions appeared a day or two later on the buttocks, legs, and thighs. The other children in the house remained well, and showed no eruption. The father had died, ten weeks before the eruption had come out on the child, of "rapid consumption." He had kept to his bed, in the same room with this child, for six weeks prior to his death. His expectoration had been very copious, and the room had not been disinfected in any way. The patient died a month later, probably of tuberculosis. A post-mortem could not be obtained. In my second case a remarkably profuse eruption of varicella had been the first departure from health. The patient, a boy of two, was admitted into the Children's Hospital with the varicella. Over two thousand lesions were counted at one time on his body, and this was so thickly covered that a penny could nowhere be laid on the surface without obscuring some lesions. This was stated to be his third

* 'Brit. Journ. of Derm.,' 1892.

* 'Path. Soc. Trans.,' vol. xxxvi, p. 471.

† 'Brit. Journ. of Derm.,' vol. xiii, p. 52.

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attack of chicken-pox in twelve months. He was discharged from the hospital after ten days, no fresh spots having appeared, but still having several pustules on his body. He was admitted again three weeks later with signs of acute miliary tuberculosis, of which he died shortly after in the hospital, and at the post-mortem all the viscera were found to be tuberculous. There were also several small ulcers and scars on the skin, and these were said to have persisted from the varicellar lesions with which he had been discharged. In both these cases sections from the pustules were examined by me histologically, and no bacilli were discovered.

Pyloric Stenosis in Infants.—E. W. Saunders ('Med. Times and Register,' July, 1902) says that in the treatment of this disease the indications are—First, the administration of some medicinal agent which shall overcome to a greater or less extent the violent contraction of the pylorus. Among the drugs to be recommended are belladonna, bromides, and chloral. Second, the treatment of the secondary gastric irritation. This results from the stagnation of food, and should be treated by washing out the stomach and by giving it rest; rectal feeding should therefore be resorted to from time to time, and for twenty-four hours nothing but water given by the mouth; when food by the mouth is again allowed, the stomach should be washed out occasionally to remove a possible residuum of undigested food. Third, the diet of the child should consist of food which forms no coagulum in the stomach. Milk or any food containing undigested casein will not answer; consequently the mother's milk is usually unsuitable, while the milk of a wet-nurse in advanced lactation will succeed. Whey or peptonised milk, or a mixture of both, is usually the best food. The deficiency in fat should be supplied by cod-liver oil. A very small percentage of cream can be gradually added. To aid the motor power of the stomach by gravity, the infant should be placed on its right side after nursing. The end to be accomplished is hypertrophy of the gastric wall without dilatation; hence the quantity of food should not be large.—*American Journ. Obstet.*, December, 1902.

THE EFFECT OF PREGNANCY ON CHRONIC HEART DISEASE.*

By CUTHBERT C. GIBBES, M.D., M.R.C.P.,

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(*Concluded from page* 202.)

Treatment.

The question of marriage in chronic heart disease has given rise to much discussion, and widely different opinions are held. It is quite possible for a woman with chronic valvular disease to pass through several pregnancies safely, and to have healthy children, but it is very difficult to decide who may and who may not marry. Each case has to be taken on its own merits. A friend of mine in general practice was recently asked to decide if a lady with mitral stenosis ought to marry, and he wanted my views on the subject. I strongly advised him to avoid giving any opinion whatever, but to find out some consultant in whom the parents especially believed, and let him decide. I find that, whatever view is taken, the medical man gets blamed. If he positively pronounces against marriage the probability is that the mother will be seriously annoyed and completely lose confidence in him. If, on the other hand, he permits the marriage to take place, he has the sole responsibility of all accidents. When compensation fails, as it will sooner or later if successive pregnancies occur, he has two or three months' abuse (silent though it may be), according to the nature of the subjective symptoms, and is blamed for the death when it occurs.

Speaking generally, the patient's position in life must rule the requirements of each individual case. Compensation that would be insufficient in one instance might be sufficient in another; therefore the capabilities of the heart-muscle require, in many instances, to be tested. An example will make my meaning plain. A porter has aortic regurgitation, and yet is able to carry heavy boxes upstairs, whereas another man runs twenty yards to catch a train and falls down dead. A woman

* Presidential Address to the Chelsea Clinical Society on October 28th, 1902.

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who has to work hard for her living all the time she is pregnant wants the same quality of muscle that enables the porter to carry the box upstairs, but those who command every luxury can safely put up with an inferior article. On the other hand, if heart-muscle will not stand the exertion of hurrying for a few yards to catch a train, it certainly will not stand pregnancy. The question of gauging the muscular powers of the heart is a very difficult one, for the ordinary methods of physical examination will not always afford us the information we require. Under these circumstances I watch the effects of exertion: first, on the size of the heart (when accurate percussion is possible); secondly, on the frequency and character of its action; and thirdly, on the respiration. Some may consider that this mode of procedure is attended with risk to the patient's life, but it must be remembered that it is only employed in compensated cases, and the patient's heart has much harder work to do if she becomes pregnant. In sanctioning marriage, it must be borne in mind that no form of heart disease will stand many pregnancies, especially if they occur in rapid succession.

Turning now to the individual valve lesions, my opinion regarding mitral insufficiency differs from many observers, and I recommend marriage more freely than in any other valvular disease. If we take the cases of mitral insufficiency recorded by Macdonald, Remy, and Porak, their aggregate deaths furnish a rate of 28 per cent. Now, presuming for the sake of argument that even one quarter of these deaths, or 7 per cent., were really due to mitral insufficiency, ought not all forms of it to be dangerous, and should we not expect pregnancy to be prejudicial to anæmic young women with mitral insufficiency, instead of improving the condition of the heart, as it generally does?

In aortic stenosis marriage should not be recommended unless we are able to satisfy ourselves that the narrowing is inconsiderable and the compensation sufficient. If the disease is of long standing, or the orifice much narrowed, marriage should be forbidden.

In aortic insufficiency there are several points to be considered before sanctioning marriage. It may be permitted when the regurgitation is not very free and arterial degeneration is absent, provided the heart is sufficiently compensated, and there is no evidence that failure has previously occurred. When the regurgitation is very free the question is much more difficult to answer, even should the arteries be sound. If there are signs of arterial degeneration, with dilatation of the aorta, or if compensation has shown signs of failing, marriage should be forbidden.

Mitral stenosis is generally considered to be the most serious form of valvular disease in pregnancy, and no doubt in a large number of instances it is so; but there are many women suffering from this disease who can marry and have one or two children with impunity. In aortic disease the condition of the left ventricle has to be especially considered, but in mitral disease the responsibility rests on the right ventricle. As it begins to fail, lung complications gradually develop, the general venous system becomes congested, and the other organs of the body suffer. The previous history of a case, therefore, furnishes us with a useful guide as to the condition of the right ventricle in the past. Marriage ought never to be sanctioned if compensation has seriously failed, and the following subjective symptoms are complained of—viz., breathlessness on any exertion, persistent cough, with occasional hæmoptysis, and swelling of the lower extremities; and if on physical examination the following conditions are present—viz. pulse quick and irregular; heart much dilated, especially the right side; presystolic murmur much shortened, and the second sound absent at, or to the right of, the apex-beat; bronchial catarrh present in both lungs, and their bases œdematous; liver much enlarged and albumen present in the urine.

The most difficult cases to decide on are those in which there is a history that compensation has failed and lung complications have occurred, but at the time of seeing the patient the compensation is effective, and no complications whatever are found. Under these circumstances it is advisable to make repeated examinations of the urine, for the condition of the kidneys is of the greatest importance when marriage is on the tapis. In one of these cases I at first found the urine normal, but on again examining it after the patient had been in town for a week, "doing" shops and theatres, a distinct trace of albumen was present, and a diminution of solids. I therefore advised against marriage, and subsequent events proved the

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correctness of my prognosis. In patients in whom compensation has never seriously failed, and where there are no indications of undue tension in the pulmonary blood-vessels, it is, as a rule, safe to sanction marriage. Even in these cases, however, it is advisable to test the condition of the heart-muscle, as it is not a very unusual occurrence for cerebral embolism to be the first indication the patient has of this disease.

When the aortic and mitral valves have been simultaneously attacked by endocarditis I always hesitate a great deal before advising marriage. The decision depends to a great degree on the extent to which the aortic valves have been involved. If regurgitation is free the heart assumes what may be termed the aortic type, the mitral valvulitis manifesting itself at first by insufficiency, and the stenosis developing only at a later period. In other cases the aortic insufficiency is slight, the chief virulence of the endocarditis having expended itself on the mitral orifice. In these cases the heart assumes the mitral type, the aortic regurgitation being insignificant, and swamped, as it were, by the mitral stenosis. We are seldom justified in sanctioning marriage under either of these conditions, but least of all when the aortic type is present. When mitral insufficiency is secondary to aortic disease marriage should be forbidden. Primary degeneration of heart-muscle and adherent pericardium also forbid marriage.

It has been stated by many writers on this subject that should signs of failing compensation show themselves during pregnancy the heart cannot improve until delivery has taken place, and if they appear before the third or fourth month the prognosis is so grave that abortion should be resorted to. There is, however, no justification for such sweeping statements. With very few exceptions the heart rapidly responds to early treatment, and it cannot be too strongly urged that the earlier in pregnancy a patient with chronic heart disease places herself under medical supervision, the better chance she has of surviving parturition and puerperium. The treatment resolves itself into an endeavour to maintain the heart-muscle in the highest condition of efficacy that is compatible with the nature of the case. On the one hand, failure of a sound muscle may be caused by want of judicious management; while on the other hand, a heart with a less perfect muscle may be safely piloted through childbirth by minimising as much as possible the special difficulties attendant on that condition. The fundamental rules are—1st. Promote the general health of the patient. 2nd. Avoid unduly taxing the heart. And 3rd. Furnish the heart with as pure a blood-supply as possible.

In compensated cases the routine of every-day life should be carried out with a view of improving the general tone of the system. In pure aortic cases the patient may almost live in the open air, but a wise discretion must be used when the mitral orifice is involved, as these patients are especially liable to lung complications. Exercise should stop short of exertion, and much stair-work should be prohibited. If the patient is in such a position of life that work is a necessity, light employment should if possible be obtained. After the seventh month the less a patient gets about the better, the reclining posture being enjoined at regular intervals during the day. Diet permits of great latitude during the early months, but care is necessary in order to avoid the dyspeptic troubles so common in pregnant women, flatulent distension of the stomach frequently proving a source of great embarrassment to the heart. During the later weeks the amount of nitrogenous food should be limited, and milk given freely. No exercise should ever be taken directly after meals. The bowels must be carefully regulated and straining avoided. In the later weeks the medical attendant should satisfy himself that the bowels are not merely opened once a day, but are sufficiently opened; castor oil being, in my opinion, the most satisfactory aperient. These measures are necessary because the retention of the fæces adds to the abdominal pressure, and any re-absorption increases the toxic character of the blood. When the mitral orifice is involved, especially if stenosis is present, chronic venous congestion impairs the functions of the abdominal organs, and may still further increase the impurities in the blood. I therefore, during the latter half of pregnancy, supplement the regular aperient with Pil. Hyd. Rhei once a week, increasing it to twice a week during the last month. In these compensated cases little, if any, other medicinal treatment is required until the later weeks. It may then be advisable in cases of mitral stenosis to assist the heart by a mixture containing the following:—Nuc. Vomic. ℥v, Sp.

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Æth. ℥xv, Sp. Amm. Arom. ℥xv, Liq. Trinitrini ℥¾ three times a day.

In aortic disease the heart not unfrequently appears to be overtaxed during the fourth or fifth month of pregnancy. I then always insist on complete rest, so as to enable the normal hypertrophy of the left ventricle to develop, and with a view to lower the arterial tension and increase the strength of the systole, I prescribe somewhat as follows:—Pot. Iod. gr. iij, Liq. Trinitrini ℥¾, Nuc. Vomic. ℥vi, Caff. Citrat. gr. ij, Sp. Amm. Arom. ℥xv, Aq. ad ℥j. T. d. s.

A month or so of this treatment will frequently so benefit the heart that more latitude may be given, and the patient be allowed to get about, continuing the medicinal treatment on the lines I have suggested.

In some cases the heart fails to respond, and dilatation of the left ventricle ensues, the result being that the mitral orifice becomes insufficient, and a fresh train of symptoms present themselves. Under these conditions absolute rest in the recumbent posture must be enjoined, and the diet must be as non-nitrogenous as the circumstances of the case permit, milk forming the principal item. An ether and ammonia mixture should always be within reach in case of syncope. Two or three doses of digitalis may be given and the effect watched, but I generally find increasing the dose of nux vomica in the mixture already given produces better results. If no benefit results, before other treatment is resorted to make sure that the drugs themselves are not to blame, for I know from personal experience that some of the tinctures of nux vomica and digitalis at present on the market are quite useless if given in the ordinary doses. Lung complications resulting from secondary mitral insufficiency do not, as a rule, assume the severe type that is met with in mitral stenosis, but they are less easily dealt with.

In the mitral type, when bronchial catarrh is present as well as œdema, the usual stimulating expectorants may be given with advantage, but no real benefit will result unless the right side of the heart is made the main object of treatment. Rest must be insisted on. The strictness with which this is enjoined depends, of course, on the severity of the symptoms, but if the lung complications are pronounced absolute rest is a necessity. This can only be obtained by confining the patient to bed, and employing a nurse or some friend to wait upon her. When this cannot be carried out the patient suffers, and the heart loses its chance of recovering compensation. I usually prescribe one sixth of a grain of calomel three or four nights running, with digitalis and strychnine in some form, and nitro-glycerine, ether, and ammonia every four hours. Larger doses of calomel may be given, but I have ceased to do so, as I get better results from the smaller ones. Digitalis is not well borne if there is much constriction, and therefore should be watched. I have tried substituting strophanthus, but I find it more satisfactory to omit digitalis occasionally and then resume it. If this line of treatment fails to relieve the symptoms, leeches should be used. I generally apply two or three down the right side of the sternum and watch the results; for more can easily be applied afterwards if required. Leeches are, I think, more efficacious in pregnant women who have mitral disease than even in the non-pregnant, and I do not hesitate to employ venesection if they fail or more immediate results are desired. I strongly advise the early adoption of prompt measures, because the heart is exposed to steadily increasing difficulties, and delay renders treatment far less effectual. Moreover fresh complications may be introduced into the case by the kidneys becoming involved. If this treatment fails to restore compensation, owing to the condition of the heart-muscle, it will certainly ameliorate the symptoms, and may prevent their increasing as pregnancy advances. In addition to the above, dry cupping over the base of the lungs will frequently relieve dyspnœa. Oxygen inhalations are also most useful; I prescribe them not only temporarily to relieve acute attacks of dyspnœa, but at regular intervals, in order to improve the condition of the heart-muscle.

Acute paroxysms of dyspnœa should be treated with oxygen inhalations and hypodermic injections of strychnine and ether, which may be repeated, if necessary, in order to tide the patient over the crisis.

Sleeplessness, if persistent, may prove most injurious to the patient, and relief should be given. If general treatment fails, I usually prescribe opium or chloretone in small doses during the day; if this also fails, a larger dose of either may be given the last thing at night.

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When albumen is present in the urine it is necessary to have a daily record made of the quantity passed, and amount of albumen present. If the albumen is due to old-standing interstitial nephritis, a non-nitrogenous diet may not be a necessity; indeed, many of these cases progress better with a more generous one. In pregnancy albuminuria the diet must also depend on the nature of the case: if there is only a trace of albumen, and the urine is normal in quantity, a purely non-nitrogenous diet is not required, as it may only be due to pressure; but if a larger quantity of albumen is present, rest and a non-nitrogenous diet should be insisted on. The gravest cases are those in which the urine becomes solid on boiling, and there is a reduction in the quantity of urine passed. Under these circumstances the patient must be kept in bed and restricted to an entire milk diet, such other treatment being adopted as the case demands.

In combined valvular lesions the treatment must depend on the orifice that is principally involved, and should be carried out in the manner I have previously indicated. Owing in a great measure to the severity of the endocarditis by which these lesions have been produced, they are liable to be complicated by an increase in the fibrous tissue of the myocardium, and our utmost endeavours are required to keep the heart going until the termination of the pregnancy. In mitral disease a fibrosed or fatty myocardium also results, sooner or later, from successive pregnancies, and the question of induction of premature labour is forced on our notice. An induced labour is more injurious to the patient than a natural one, and we have to decide which of the two will give the patient the best possible chance of life, for the risks to the child's life being about equal, the mother's life ought to be the first consideration. It has frequently been used as an argument against premature induction that heart failure is equally frequent during puerperium whether labour is artificial or natural. This is no doubt true if induction has been postponed until Nature almost saves the medical attendant the trouble, but it is not true when induction is undertaken before urgent cardiac symptoms arise. The majority of cases are better left alone, but if premature labour has to be induced it should be done before the cardiac symptoms become urgent and the patient is worn out by the long struggle. If this opportunity is lost, the patient stands a better chance if left to Nature, the probability being premature labour will come on by itself. In cases of hydramnios, when the upward displacement of the diaphragm largely increases the urgency of the symptoms, labour should be always induced.

Abortion is a much more serious matter, the danger to life being greater than in induced or natural labours. It is only justifiable when we are satisfied that the probabilities are against the patient living until premature labour can be induced. If we have a history of severe heart failure with pronounced kidney complications during the last pregnancy, and we find during the early months of the present one that compensation is failing and albumen has reappeared in large quantities in the urine, abortion should certainly be procured.

During labour the heart should be relieved of as much strain as possible. In the first stage bearing down should be avoided, and the patient should be allowed to assume whatever position affords her the most comfort; when the breathing is much embarrassed oxygen inhalations should be administered from time to time. If the heart shows any signs of flagging, ether or strychnine should be administered alternately. Artificial dilatation of the os must be resorted to if necessary, and in any case an anæsthetic should be administered and the forceps used as soon as the os is fully dilated. Phillips recommends that the "extraction of the fœtus should be performed slowly and a twelve-pound sand-bag laid over the fundus uteri during the process (the patient being in the dorsal decubitus), so that its pressure may counteract the rapidly descending abdominal pressure." I am aware that I am treading on dangerous ground in differing from an obstetrician, but I cannot help thinking that the first effect of diminished abdominal pressure would be to relieve the over-distended right ventricle, and any post-partum abdominal pressure must defeat the object we have in view.

It is therefore never advisable to hurry the removal of the placenta, but rather to allow free hæmorrhage to take place, for these cases die far oftener from too little hæmorrhage than from too much. Sudden death from over-distension of the right ventricle is especially liable to occur at the

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termination of labour or shortly after, and we must be prepared to adopt prompt measures. Fraser Wright has strongly recommended the use of nitrite of amyl under these circumstances, in order to relieve the heart by "dilatation of the peripheral systemic arterioles." In some cases this mode of treatment has saved life, and it should always be within reach, for the right ventricle may fail from over-distension before there is time for venesection to be performed, or any other treatment to be carried out. If it affords relief it does not contra-indicate venesection, but rather gives the medical attendant sufficient time to perform it, hypodermic injection of strychnine and ether being also given. Digitalis is most useful later on, but under the conditions above mentioned more benefit will, I believe, be derived from strychnine. In cases where the patient survives parturition but fails to rally, strychnine, digitalis, and ether should be pushed, and, if the symptoms point to distension of the heart, leeches should be applied or venesection adopted as the symptoms indicate. Absolute rest in the horizontal posture should be insisted on for at least the first week, and the patient should not be allowed to sit up for three or four weeks, according to the nature of the case.

Lactation is generally forbidden because it tends to keep up the cardiac hypertrophy (Macdonald) and weakens the patient. I cannot altogether agree with this, for in some aortic cases I have found it decidedly beneficial if continued only for a few weeks.

In conclusion, gentlemen, I would say: Take your time before advising any woman with heart disease to marry, but when pregnancy occurs, find out as much as you can about the heart before compensation fails, and if it does, act promptly.

Poisoning with the Douglas Pine. — A woman, sixty years of age, was occupied in making decorations with branches of pine. After two days of this work she was taken sick with nausea and headache, followed by a stuporous condition with difficult breathing and some delirium. She scarcely responded to even loud cries. The condition continued with gradual improvement up to a month after the onset of the attack. A month later she was practically well.— *Philadelphia Med. Journ.*, December 20th, 1902.

SELECTED ESSAYS AND ADDRESSES, by SIR JAMES PAGET. Edited by Stephen Paget, F.R.C.S. (Longmans, Green and Co., 1902.)

There is a singular charm about these essays and addresses of Sir James Paget, a charm which is due in part to the style of writing, and in part to the knowledge and thought which are manifested in every page. To those practitioners who have not advanced far on their career, and who intend to write medical papers, we should commend these essays as models of style. Whether he was relating the life of John Hunter, or describing the characteristics of osteitis deformans, or addressing the International Medical Congress, Sir James spoke and wrote in a manner which is now but rarely met with, even amongst the leaders of the profession. At times his form of expression becomes almost Biblical, as in the following sentence:—"I had a boy aged about fifteen in the hospital who had these night pains in a most marked degree in one knee, and the joint was a little swollen; and he, being tuberculous, was wasting, hectic, and very ill." One is struck by the careful clinical observation and the large experience of cases which are represented by papers such as those on "Nervous Mimicry." They form a useful and a permanent addition to medical knowledge, for he does not discuss transient theories or speculative pathology, but gives a full account of the various phenomena present, and the differential diagnosis from forms of organic disease. Other chapters are devoted to subjects which are not, as a rule, taught to medical students, but which are of extreme importance in connection with private practice. The family practitioner is daily confronted with problems, human problems, which are not solved for him by the text-books, but which will be found treated with sound common sense in this volume. Those who follow the author into the by-paths of medicine, which he sometimes pursues, as in the panegyric on John Hunter, the history of anæsthetics, and the paper on experiments on animals, will find him a pleasant and persuasive guide. His latest papers, written after he had retired from active practice, do not appear to have quite the same value as those of his earlier years, but they are well worthy of a place in this memorial of Sir James Paget's life and work.

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* *Specially reported for The Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE

ON THE

TOPOGRAPHICAL DIAGNOSIS OF PARALYSIS.

Delivered at the Polyclinic, November 12th, 1902.

By JUDSON S. BURY, M.D.(Lond.), F.R.C.P.,

Physician to the Manchester Royal Infirmary.

LADIES AND GENTLEMEN,—My subject to-day is the topographical diagnosis of paralysis,—that is to say, the regional diagnosis of paralysis. I am not going to refer, except perhaps incidentally, to the nature of the disease; I shall limit myself entirely to the site of the lesion.

Now it is obvious that loss or impairment of voluntary power to move a part of the body must depend either on disease of the muscles which are necessary for the movement, or on interruption of the nerve-impulses which normally are able to stimulate the muscles to action. At the outset, then, two classes of paralysis are indicated: first, a class in which disease of muscular tissue is the essential feature; and, secondly, a class in which the paralysis depends on interruption of nerve-impulses—that is, on disease of the nervous system itself. To fully understand this second class, we require to have an accurate knowledge of the paths by which nerve-impulses are conveyed to the muscles. This knowledge is now pretty definite and complete, and it will be well to briefly consider the chief points connected with the anatomy of the motor path which are helpful to us in determining the position of the lesion in different cases of paralysis.

In all probability the psychomotor region of the cortex occupies a smaller area than was formerly supposed—the older view is represented in the diagrams, where you see that the motor centres occupy the ascending frontal and ascending parietal convolutions, together with the adjacent portion of the superior parietal lobule and some portion of

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the median surface of the hemisphere. Recent experiments, however, by Professor Sherrington indicate that all motor functions are related to the ascending frontal convolution, and that the ascending parietal convolution does not form any part of the motor area. In this connection it is interesting to note that the large pyramidal nerve-cells which are so abundant in the ascending frontal are not found in the ascending parietal convolution. The axis-cylinders of these pyramidal (or motor, as they may be called for convenience) cells are prolonged into fibres which constitute the motor path, the situation of which is well shown in the diagrams and in the beautiful models kindly lent by Professor Stirling. You will see that the fibres pass through the white substance of the hemisphere, converging to the internal capsule, where they occupy its "knee" and the anterior two-thirds of its posterior segment. The leg fibres are the furthest back, and are next to the sensory tract. From the internal capsule they pass to the crus, where they occupy the middle two-fifths of the crusta. We come next to the pons, where (as the diagrams show) the fibres for the face and tongue cross the middle line to reach the nuclei of the facial and hypoglossal nerves. In the pons the limb and trunk fibres lie between the superficial and deep transverse fibres; they then pass into the medulla, where they constitute the anterior pyramid, the greater proportion of its fibres crossing over at the decussation to run in the lateral column of the cord and form the "crossed pyramidal tract." A small though variable proportion of fibres is continued from the anterior pyramid of the medulla into the anterior column of the same side of the cord, and probably also a few into the lateral tract of the same side.

The fibres of the pyramidal tract end in grey matter near the motor cells in the anterior horn, and from these ganglionic cells spring the anterior roots, which are composed of motor fibres destined for the muscles. In a similar manner the tracts for the motor cranial nerves separate from the pyramidal tract in the crus, pons, and medulla; they cross the middle line and end near the ganglionic cells which constitute their nuclei in the neighbourhood of the fourth ventricle, and from these cells issue fibres which are collected together to form the motor cranial nerves.

The cortical motor areas are the centres for voluntary movement. The anterior horns of the cord and the corresponding nuclei in the pons and medulla transmit voluntary motor impulses to the peripheral nerves. The cortical centres preside over the nutrition of the pyramidal tract, the bulbar and spinal nuclei over that of the motor fibres in the peripheral nerves.

There is thus a natural division of the motor path into two parts, and we shall presently find that this division is very convenient when regarded from the clinical standpoint.

The upper part, as we have seen, extends from the cortical centres along the pyramidal tract to the bulbar nuclei and to the anterior horns of the spinal cord; while the lower part includes the bulbar nuclei and the anterior horns, together with the motor fibres which extend from them to the muscles. These parts are now usually called central and peripheral neurons—that is, the upper segment of the motor path is composed of central neurons, the lower segment of peripheral neurons—a neuron meaning a cell-body with its processes, the chief or axis-cylinder process being called the axon, the branching processes dendrons. This division of the motor path into two parts corresponds, as I have just suggested, to a convenient clinical classification of cases of paralysis. For we find that when the upper neurons are affected paralysis is associated with spasm or rigidity of muscular tissue, whereas when the lower neurons are affected paralysis is associated with flaccidity and wasting of the muscles. We have, then, two great groups of paralyses—namely, the spastic and the atrophic; the former indicating impairment of the functions of the upper neurons, the latter of the lower neurons. A third group of paralyses is constituted by cases in which an atrophic and a spastic paralysis are found combined in the same patient. This may be designated the group of mixed paralyses.

Spastic Paralysis.—Let us first consider the two simple groups, beginning with the spastic one. In this variety the muscles maintain their normal bulk, but their tonicity is increased and the affected limbs present varying degrees of rigidity. The excess of tonus may be so trifling as only to show itself by a very slight increase of resistance to passive movements, or possibly merely by an exaggeration of the deep reflexes (for these are closely connected with the tone of the muscles); or it may be so great that the muscles feel as hard as boards

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even when the limb is at rest, so that movement of it by the antagonistic muscles is impossible.

Another peculiarity of spastic paralysis is that the paralysis is diffuse—that is, all the muscles of a limb, or of a portion of a limb, are paralysed; and although some muscles are weaker than others, it is rare for any muscle situated within the affected region to escape from some degree of paralysis. In this respect it differs from atrophic paralysis, which for the most part picks out particular muscles or groups of muscles. A spastic paralysis is evidence, then, that some part of the upper neurons has its functions impaired or abolished, and I would here remark that the condition of the affected part may be the same whether the lesion be situated in the motor cells of the cortex, in the pyramidal fibres, or in their terminal ramifications in the grey matter of the cord. To determine the locality of the lesion we have to consider the distribution of the paralysis together with the presence or absence of sensory and other associated symptoms. Spastic paralysis may affect one side of the body, one limb or both legs—that is, it may be hemiplegic, monoplegic, or paraplegic in distribution.

As an illustration of the method referred to for investigating a case of paralysis, let us take an ordinary case of hemiplegia, where the face, arm, and leg are paralysed on the same side of the body. We find that the arm and the leg are in a state of rigidity, and we say at once that the upper neurons must be affected. Where is the lesion likely to be? Our knowledge of the motor path tells us at once that it must be above the middle of the pons. For if you draw a line across the middle of the pons you see that the fibres from the facial nucleus cross over at the spot I have indicated, and you also see that the limb fibres have already crossed at the lower part of the medulla. Hence, if you trace these fibres up it is clear that the lesion must be on the opposite side of the brain, whether in the upper part of the pons, or in the crus, or in some part of the cerebral hemisphere. Is the lesion likely to be in the crus? There the fibres are close together, but you see in the diagram the close relations of the crus to the third nerve; hence we should naturally expect and certainly do find in practice that as a rule in lesions of the crus we have, in addition to the ordinary type of hemiplegia, paralysis of the eye muscles supplied by the third nerve on the same side as the lesion. Thus we have ptosis, and on raising the lid we note an external squint and an inability to move the eyeball upwards, downwards, and inwards. If there is no eye paralysis we are limited to a consideration of the internal capsule and the hemisphere above it. Now obviously, where the motor fibres are close together, as in the internal capsule, is the most likely place; and this we find to be the case in actual practice, and that hemiplegia becomes rarer the nearer the lesion is to the cortex—that is, the more divergent the fibres become. As we get nearer to the cortex we are more likely to get a monoplegia, one limb or one side of the face being affected. When the cortex itself is diseased clonic spasms often attack the paralysed side, because there is often irritation of cortical cells which are not completely destroyed by the lesion.

In the case of a monoplegia errors in diagnosis will often be avoided by remembering the features of the classification I have sketched. For example, a man consulted me for partial paralysis of the left arm; the muscles were not wasted and there was no distinct spasm or rigidity of the limb. Was the upper or lower neuron involved? He complained of numbness in the fingers, and on testing the skin partial anæsthesia was discovered. The case looked like one of neuritis affecting some of the nerves of the brachial plexus. But on testing the wrist-jerks, the left wrist-jerk was much more active than the right one. This was taken as evidence that the tonicity of the muscles was increased, and that the upper rather than the lower neuron was involved. Hence it was thought probable that even in the absence of headache and optic neuritis there might be a tumour implicating the arm centre in the cortex. Subsequently this diagnosis became clear, and the skull was trephined and a tumour found.

Let us glance now at cases of spastic paraplegia. Here, in a typical case, both legs show a varying degree of paralysis combined with decided rigidity; the knee-jerks are exaggerated, and ankle-clonus is easily obtained. We know at once that the upper neurons for the legs are involved. These, as we have seen, extend from the cortices close to the longitudinal fissure down to the lumbar portion of the spinal cord. Hence the lesion may be in the brain or in the cord. A mere consideration of the spastic paraplegia by itself would not enable us to decide between the brain

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and the cord. We deem it likely on anatomical grounds that the lesion will be where the pyramidal tracts are near together, that is in the cord, or possibly in the medulla or pons. The next most likely place would be the cortex, because there the centres for the legs are close together. These considerations are borne out in practice. We find that in the child the lesion may be in either place, while in the adult it is nearly always in the cord. The diagnosis, as a rule, is easily made by the presence of other symptoms, but I wish to insist on the similarity in the conditions of the legs whether the brain or the cord is affected.

If, then, we meet with *spastic paraplegia in the child* we must always think of the cortex as well as of the spinal cord. If the paralysis is associated with sensory symptoms and with bladder disturbance, then it is dependent on spinal myelitis, which, in most cases in the child, is started by caries of the vertebræ. But if unassociated with other symptoms—that is, if it is a pure spastic paraplegia—it is nearly always due to cortical disease, and two classes of cases may be distinguished:

(1) Cases known as "birth palsies," where either a meningeal hæmorrhage at birth has led to cortical degeneration or the latter has occurred independently; and (2) cases of hereditary syphilis, where a meningo-encephalitis occurring in early life has produced sclerosis of the cortex with resulting degeneration of the pyramidal tracts. In both classes—owing to the cortical disease, which is rarely limited to the leg centres—the child's intelligence as a rule is impaired; the mental backwardness may be slight in degree or may amount to profound dementia. In both classes, too, convulsive attacks may occur, while we often find some evidence of spasm in the hands. A diagnosis between the two classes is obviously made by the presence or the absence of the well-known signs of late hereditary syphilis, for the discovery of which we are so much indebted to our friend Mr. Hutchinson. I allude especially to the upper central incisor teeth, which, in typical cases, are notched and pegged. But typical teeth are not always present, and it is important to examine the upper central incisors very carefully in order to detect slight notching and slight narrowing of the cutting edge. Other signs of hereditary syphilis must also be looked for—namely, symmetrical keratitis, scars at the angles of the mouth, deafness, etc. It is interesting to contrast this type of brain disease with that due to acquired syphilis. The latter type is a hemiplegia due to central softening from endarteritis and thrombosis, or a monoplegia with unilateral convulsions from a gumma in the cortex.

We may now pass to the diagnosis of spastic paraplegia which has developed in *adult* life. Here, as a rule, we have only to consider affections of the spinal cord. Again, we begin by saying that the upper neurons, that is the pyramidal tracts, are affected, and we try to ascertain whether they are alone affected or whether other elements of the cord are also involved. In the latter case we should expect to find sensory symptoms and disturbance of the bladder and rectal functions. We should first ask ourselves—is there any evidence of disease outside the cord? This would be shown by signs indicating that the spinal roots were involved. Thus irritation of the posterior roots would give rise to pains round the body or down the limbs, while degeneration of the anterior roots in the cervical region would lead to muscular atrophy in the hand or arm. Then, too, an examination of the spinal column might reveal an angular projection, as in caries, or local tenderness to pressure without any obvious deformity. If we can exclude caries, pachymeningitis, and other diseases beginning outside the cord, then we must consider diseases affecting the cord alone. The common condition is some form of myelitis above the lumbar region. The height of the lesion is determined by the upward extent of paralysis and of anæsthesia, while the condition of the superficial reflexes often gives indications with regard to the vertical downward extent of the lesion.

But supposing we have to deal with an uncomplicated spastic paraplegia. This must be due to disease of the pyramidal tracts alone, and, at any rate, there is no evidence that other parts are also affected. Such a condition has been described as "primary lateral sclerosis." I think it is questionable whether such a disease ever occurs. Certainly the large majority of cases of pure spastic paraplegia in the adult turn out to be instances of disseminated sclerosis—that is, the pyramidal tracts are affected first, and subsequently other parts of the nervous system become involved, as is shown by the presence at a later period of the disease of nystagmus, tremor, and scanning speech.

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But these classical symptoms may never appear, or only appear at a very late period. And even in the uncomplicated cases of spastic paraplegia—the so-called primary lateral sclerosis—a careful examination will often reveal indications that the grey matter of the cord as well as the pyramidal tract is affected; thus the superficial abdominal reflexes may be diminished or absent on one side or on both sides of the body. My experience teaches me that uncomplicated spastic paraplegia is an important symptom of disseminated sclerosis, and should receive a more prominent place in descriptions of the disease than is usually to be found in text-books on medicine.

Let me repeat, an uncomplicated spastic paraplegia should suggest in the child cortical disease, in the adult disseminated sclerosis.

Atrophic Paralysis.—Turning now to atrophic paralysis, we find that muscular weakness is associated with muscular atrophy. Such a condition indicates one of two things, either (1) impaired or abolished function of some part of the lower motor neurons, or (2) disease within the muscles themselves. In the former case the atrophy is sometimes termed degenerative or spinoneural; in the latter primary myopathic.

The diagnosis of a case of atrophic paralysis is based partly on its distribution and partly on the presence or absence of sensory and other symptoms. When we find weakness and wasting of a single muscle, or a group of muscles supplied by one nerve, then we assume that the lesion is situated in the nerve or in its nucleus; for example, paralysis of the facial muscles from a lesion of the facial nerve or its nucleus, paralysis of the deltoid from a lesion of the circumflex nerve, or paralysis of the flexors of the fingers and of the interossei from a lesion of the ulnar nerve. In the two last cases sensory symptoms may also be present, for the circumflex and ulnar nerves contain both sensory and motor fibres. When a group of muscles which act in functional association, but which are supplied by different nerves, become paralysed, we know that there is a lesion of the anterior horns or of certain of the anterior roots, or possibly of the nerve plexus which supplies the affected limb; for example, paralysis of the deltoid, biceps, and supinator longus, from a lesion of the fifth cervical root, or of the upper portion of the cervical enlargement. If the atrophic paralysis depends on disease of the fifth root, we should also expect a band of anæsthesia down the outer side of the arm as far as the styloid process of the radius. Of other groups of muscles we may mention those which are affected in infantile paralysis. One common type is an atrophic paralysis of the muscles on the front and outer side of the leg; another type is that in which the deltoid and other shoulder muscles are affected. Infantile paralysis is an example of atrophic paralysis uncomplicated by sensory or other symptoms, and, as we know, is due to a lesion of the cells of the anterior horn. As a rule, only one limb is involved; occasionally two limbs, or even all four, may be affected. When both legs are affected we have an example of atrophic paraplegia, but this more commonly depends on a lumbar myelitis, and then is associated with incontinence of urine and with anæsthesia in the territory of the nerves supplied by the sacral or lumbar plexuses.

We may now briefly glance at three types of wide-spread atrophic paralysis—namely (1) multiple neuritis, (2) progressive muscular atrophy or chronic anterior poliomyelitis, and (3) idiopathic muscular atrophy.

The first variety is due to a degeneration of a number of the peripheral nerves, the second to a slowly progressive atrophy of the nerve-cells in the anterior horns, and the third to decay of the muscular tissue itself.

The symptoms of multiple neuritis were fully discussed in my lecture of yesterday; it must suffice to say that the most characteristic feature of the disease is symmetrical localisation of motor and sensory symptoms in the peripheral parts of the limb. The chief motor symptom is weakness of the extensor muscles of the hands and of the flexors of the ankles, giving rise in typical cases to double wrist- and double foot-drop; while the sensory symptoms comprise diminution and perversion of cutaneous sensibility, together with tenderness of the nerve trunks, and especially of the muscles. The presence of sensory symptoms is sufficient to distinguish the disease from the other two varieties of muscular atrophy. What are the distinctions between progressive and idiopathic muscular atrophy?

First of all the distribution of the atrophy is usually different in the two diseases. Thus in progressive muscular atrophy the small muscles of the

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hand are the first to be affected, so that the thenar and hypothenar eminences become flat, and hollows appear between the metacarpal bones. At first one hand only is affected, but soon the muscles of the other hand begin to waste. Then the muscles of the forearm are attacked, and often at a later period the muscles of the shoulder girdle. But in the idiopathic variety the hand muscles and the forearm muscles, with the exception of the supinator longus, are spared, while those of the upper arm, the shoulder, the thigh, and occasionally those of the face, are affected. Sometimes progressive muscular atrophy begins in the shoulder muscles, and then a difficulty in diagnosis may be experienced. But here also a difference in distribution may be noted. Thus in chronic anterior poliomyelitis the latissimus dorsi, the lower half of the pectoralis major, and the triceps often entirely escape, whereas they are often markedly atrophied in the idiopathic type. Moreover the platysma, the levator anguli scapulæ, and the upper part of the trapezius are spared in anterior poliomyelitis, but are frequently affected in idiopathic muscular atrophy. The former disease is also distinguished by the presence of fibrillar tremors in the muscles and by the reaction of degeneration which they give to electricity. I would draw your attention also to the presence of enlargement and hardening of some part of a muscle in idiopathic muscular atrophy; this may often be seen in the lower part of the deltoid, while its upper portion is much wasted. Indeed, if cases of this disease be carefully studied, it will be found that some present enlargement of several muscles and that there is no abrupt limit between the disease and pseudo-hypertrophic paralysis, where enlarged hard muscles are the prominent features.

There are some other cases of muscular atrophy which it is interesting to notice, because they show the relation and the influence of one neuron on another. *Arthritic muscular atrophy* is a case in point. When a joint is injured or diseased the muscles that move the joint, and especially the extensor muscles, undergo atrophy. Thus if the shoulder is affected the deltoid chiefly wastes; if the knee, the muscles in front of the thigh. Charcot's explanation is probably the best one—namely, that the nutrition of the motor cells of the cord is deranged in a reflex manner, morbid impulses being conveyed to them by the sensory nerves from the joint, and that this derangement or torpor determines the wasting of the muscles. In this connection I may mention that it has been shown experimentally that a lesion of the posterior roots will occasionally lead to wasting of some of the cells in the anterior horns. Again, there is the *hypotonus* of the muscles in locomotor-ataxy, which is another illustration of the influence of sensory on motor neurons. In tabes, sometimes at an early period of the disease, the leg, owing to the looseness of its muscles or their lowered tonus, can often be raised to an angle of 90 or more degrees from the couch.

Now in practice we frequently meet with cases of muscular atrophy which are difficult to account for, and I think we are often much helped in diagnosis by remembering the possibility that irritation of sensory nerve-fibres has an important influence on the nutritive condition of motor neurons. I believe, indeed, that progressive muscular atrophy is sometimes started in this way. We may also consider the influence of the terminals of the pyramidal fibres on the motor cells of the cord—that is, the influence of the upper on the lower motor neurons. Thus very rarely in hemiplegia muscular atrophy may occur when no detectable changes can be found in any part of the lower neurons.

The above examples, then, may be taken as indications that the vitality of nerve-cells depends to some extent on the nature of the sensory and other impulses which are brought to bear on them by adjacent neurons. I wish to lay stress on these considerations, because I am quite sure they have an important practical bearing in the investigation of disease of the nervous system.

So far, I have mainly dealt with more or less typical cases of muscular atrophy where there is but little difficulty in deciding which part of the neuron is affected—that is, whether the case is one of neuritis, or anterior poliomyelitis, or whether it is one of myopathic atrophy. But occasionally we meet with generalised forms of muscular atrophy, as a result, for example, of lead poisoning or of rheumatism, in which the regional diagnosis is very difficult and often impossible. If the atrophic paralysis is ushered in by pains and other sensory symptoms we have suggestions that the peripheral nerves are at fault; but cases occur where sensory disorders are entirely absent, and

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where the lesions have been limited to the peripheral nerves. I think we must, indeed, recognise that occasionally a wide-spread atrophic paralysis of purely motor type is due to an affection of the motor fibres of the peripheral nerves. Why, indeed, should this be regarded as more remarkable than the picking out of a small group of nerve-cells or of other individual nerve elements of the spinal cord?

Let us, at any rate, continue to patiently record the clinical and pathological features of cases that come before us, and if we make deductions from them let us try to make sure that our deductions are warranted by the facts, and are not warped by a previous mental attitude towards them. It is our too easy acceptance of authoritative statements regarding disease, and the mental bias that results therefrom, which prevent us seeing things clearly, and thus tend so much to hinder the advance of scientific medicine.

THREE LECTURES

ON

COMPLICATED FRACTURES AND THEIR TREATMENT.

Delivered at the Medical Graduates' College and Polyclinic, March, 1902.

By C. B. KEETLEY, F.R.C.S.,

Senior Surgeon to the West London Hospital.

LECTURE II.

Dressing.—There are many methods of dressing—antiseptic, aseptic, and mixed, involving the use of various materials and of a great variety of drugs. The particular choice of these is, in my opinion, less important than that the surgeon should master one plan, and, having found it effectual, stick to it.

For many years I have adhered to the following:

Materials.—Iodoform gauze (10 per cent.), double cyanide gauze (3 per cent.), salicylic wool (4 per cent.), unmedicated absorbent wool, wood-wool pads, common brown strapping, bandages, as well as materials for boracic fomentations. The iodoform gauze and the double cyanide gauze are kept wet with 1 in 20 carbolic in stoppered bottles if the gauze is not going to be used up quickly, otherwise rolled up in red jaconet. For the pads the wood-wool is pulled into small pieces, placed in bags of carbolised gauze, and rolled flat after the method used by Prof. Macewen. The advantage of the carbolised gauze for this purpose lies in its stickiness. Common brown strapping and yellow diachylon are free from the irritating blistering properties of some of the plasters sold on reels.

The process of dressing.—The gauze used is carefully rinsed and squeezed out in 1 in 2000 warm sublimate lotion, and first a single layer placed on the wound. An old house surgeon of mine, Dr. Cecil Banting, pointed out to me that this is as good as a protective, greatly facilitating the ultimate removal of the dressing. On the top of this single layer goes a mass of gauze. For sound, reliable work in a serious case, no other gauze is equal to iodoform. It will keep discharges sweet much longer than will double cyanide.

Iodoform eczema is mainly a bogey created by the practice of sprinkling iodoform crystals or powder on the skin (a thing I never do), or by excessive use of strong lotions in conjunction with iodoform. What can anyone expect but eczema, or at least erythema, who uses 1 in 20 carbolic lotion without carefully washing it off, *plus* iodoform powder or *plus* gauze which he has never rinsed out? I notice that my new house surgeons tend to blame the iodoform for the results either of too much use of strong carbolic lotion or of septic causes. Such septic influences most commonly come into play in connection with wounds covered by plaster-of-Paris cases, and forgotten or neglected. Wood-wool pads are especially useful when discharge is likely to be profuse. When aseptic discharge dries they cake and mould into firm supports, acting as a splint as well as a dressing. Salicylic and absorbent wool should generally be used extravagantly, thereby adding to the comfort of the patient. A layer of plain wool wrapped quickly round a salicylic wool dressing adds to the comfort of the surgeon by saving much sneezing.

Bandaging.—When there is any likelihood of recurrent hæmorrhage, or of considerable oozing, *e. g.* after the use of Esmarch's bandage, the dressings should be bandaged on firmly. I scarcely

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ever remove the Esmarch's tourniquet before bandaging on the dressings.

If there is no likelihood of recurrent hæmorrhage, bandaging loosely will cause the patient less pain. When there is no fear of bleeding, and a many-tailed bandage will suffice to secure the dressings, it should be preferred, as its removal and re-application do not disturb the fragments.

Strapping.—A few strips judiciously arranged make the dressings snug. Non-irritating strapping, spread on brown holland or on sailcloth, is required for extension purposes. Avoid varieties which have an aromatic smell, as they often blister.

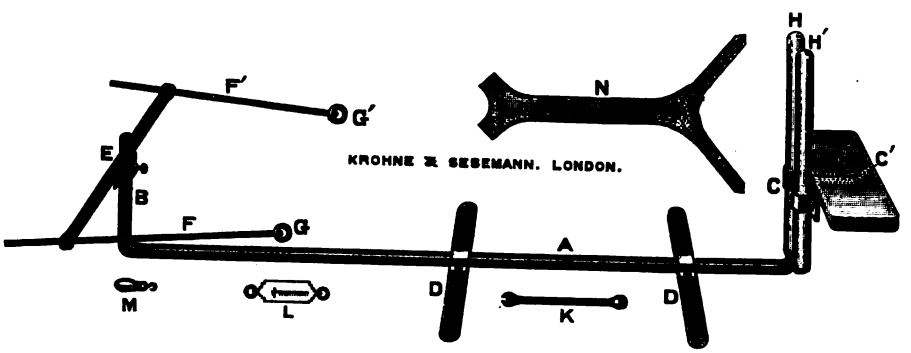
Boracic acid fomentations should be made in the orthodox way, *i. e.* of boracic lint wrung out in boiling or nearly boiling water, and well overlapped with jaconet or oil-silk. Common lint soaked in weak boracic lotion is a poor substitute. *For bandaging the head*, carbolised gauze fits better, keeps in place better, and looks much better than white cotton or linen bandages.

Fixation by external means.—Those chiefly in use may be classified as (1) movable splints; (2) so-called "immovable" appliances, especially plaster of Paris; (3) weight-extension: and combinations of these may be employed.

For keeping up correct position and full extension, in the great majority of cases no other external applications are equal to plaster of Paris. But its application takes time, its successful use requires experience, and it is quite inapplicable when there are wounds situated where the plaster case would press, *e. g.* over the malleoli in fractures at or near the ankle. I have described in great detail the rules for working a good plaster-of-Paris case in my 'Orthopædic Surgery,' chap. xviii, p. 484.

Movable apparatus, such as wooden splints, with and without iron segments, iron splints, wire splints, zinc splints, are familiar to you; and I need not add that they vary in size and shape with the part of the body for which they are intended.

Whatever splints or appliances may be used for a case of compound fracture, they should be so arranged and fixed that, if the wound should require frequent dressing, the change of dressing can be effected without removing the splints, or otherwise disturbing the bones; *e. g.* if there be a wound just above the ankle, the appliance should fix the foot, and the upper two thirds of the leg as well as the knee firmly, while at the same time it leaves the wound and the skin for some distance around it quite free. If my L-shaped pins be used, they should be put in from a side different from that on which the wound lies. As I have already said, for any wound requiring frequent dressing, a many-tailed bandage is much less disturbing than a roller.



Extension involves counter-extension. And *counter-extension* is sought for either by (1) the use of loops such as the well-known perinæal band, or (2) the utilisation of the weight of the patient's body or of a part of it. Neither method is ideal. There can be no perfect fixation when either of these two methods of counter-extension is used. The combination of full extension with fixation can only be achieved by first fully extending the fracture, and then applying a perfectly moulded and rigid cover (usually of plaster of Paris), extending up and down the limb as far as and slightly beyond prominent bony parts which cannot be approximated without the fragments overlapping, *e. g.* the bony prominence of the knee and of the ankle in fractures of the leg.

I will now describe and exhibit an extension frame which I have found useful in setting and fixing both simple and compound fractures.

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A is a bar of Bessemer steel, bent up at right angles at the two ends. It is laid upon the operating table with the end towards B, projecting beyond the foot of the table. The end C then reaches the middle of the table, as the horizontal part A is about 4½ feet long. D and D are flat pieces of steel, which, when placed as in the drawing, keep the apparatus from falling over. Each turns on a pivot for purposes of packing. Each also slides up and down the bar A. The horizontal bar E slips over the upright B, and is fixed by a small screw. At each end of E is a hole through which the long screw F (or F′) works, when it is turned by the key K. G G′ are pivoted rings. H H′ are upright wooden pins placed a quarter of an inch apart. J is also of wood, and slips on or off C′, which is one end of the bar A, greatly flattened and bent at the point C to the horizontal again. N is one of two leather anklets. L represents a dynamometer, and M a pulley.

Suppose the frame is to be used for a fracture of the shaft of the femur of an adult. Pillows, cushions, or rolled blankets are placed on the table to the height of the board J. Cotton wool is wrapped round the pins H H′, and also placed on the board J. The wool is bound in position with a bandage. If the hip is to be put into plaster of Paris the board J is removed, and the iron C wrapped in wool serves to support the patient's sacrum.

The patient is lifted into position so that the pins H H′ lie between his thighs, one on each side the middle line of the perinæum. (The object of having *two* pins is to protect the urethra from pressure.) The ankles are now padded with wool, and one of the two anklets N is placed on each ankle, the straps adjusted and buckled. By means of stout cord the anklets are connected with the rings G G′ respectively, and the dynamometer L is used on the side of the fracture. The use of the dynamometer should never be omitted, as the appliance is strong enough to pull a limb off. If greater force than the dynamometer will measure is required, the pulley M or a pair of pulleys should be used, the dynamometer being then attached to the horizontal bar E instead of to the ring G or G′. The use of the pulleys has certain inconveniences, because a part of the length of the long screw F is consumed in taking up the slack of the cord, and unless the combination is arranged carefully, and the cord fixed skilfully, scarcely enough of F may be left to extend the fracture. Of course this difficulty could be easily and simply provided against by lengthening the bar A (or by making it telescopic); but it is desirable to keep the appliance simple and of moderate size.

The cord and an iron stirrup, which fits into the leather loop N, do not appear in the drawing.

An *anæsthetic* is now given if necessary.

Then extension is made slowly on the fractured side. The other limb should have been extended, and the perinæum brought right down to the pins H H′ before commencing with the anæsthetic.

The two limbs should be carefully compared by measurement with a tape during the operation. Inquiry should have been made beforehand if any difference existed prior to the accident. One or other limb may have been broken before.

When extension is complete the next procedure depends on what is to be done.

If plaster of Paris is to be applied, the cushions beneath the limb are pulled away, leaving the limb stretched free, like a telegraph wire, between the uprights C and E.

But if pins, screws, or wires be used, the cushions should be left in place during the operation. Sand pillows, or at least one sand pillow, should be employed in this case.

When the plaster of Paris is set, the patient is set free by taking off the anklet, after releasing the screws F F′, and if the femur be fractured, by then pulling the frame downwards until the iron J slips out of the pelvic part of the plaster case. The plaster should previously be well pressed in below the anterior superior iliac spine. Its upper edge towards the perinæum should have been thickly padded with wool and several layers of flannel bandage.

When the *fracture* is *of the leg bones*, the leather anklets may have to be left beneath the plaster. They are not, however, very expensive articles, and will be available again when the plaster case is taken off.

CUSHING ('Jour. Nervous and Mental Disease,' Nov., 1902) reports a case of Raynaud's disease in which the symptoms were severe, the arterial spasm pronounced and associated with pain. For several months a condition of local asphyxia of all the digits had been present with exacerbations of almost daily occurrence. On several occasions these attacks were so severe that slight superficial patches of terminal gangrene had affected the pads on one or more of the fingers or toes. The tourniquet was used and gave very satisfactory results. —*Philadelphia Med. Journ.*, Dec. 27.

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WITH DR. E. W. AINLEY WALKER AT GUY'S HOSPITAL.

GENERAL PATHOLOGY OF INFECTION—IV.

LAST lecture we discussed the formation and the action of antitoxins; we must now pass to the consideration of the anti-microbic processes within the living body.

In this relation we must first examine the function of the phagocytic cells. Here we are indebted first and chiefly to the laborious and distinguished work of Metchnikoff. This masterly observer, following the guidance of his own discoveries in comparative pathology and physiology, came, as we saw, to look upon the phagocytes as the essential agents in protection against living micro-organisms.

But his views received by no means general acceptation, and where his observations were not open to question, yet his interpretation of them at least was strongly combated.

Out of this opposition to the purely cellular theory of anti-bacterial action, as first conceived by Metchnikoff, there arose the humoro-cellular theory of which I spoke to you; this, in its turn, reacted on and has considerably modified the views of Metchnikoff himself. It maintains that at least a part of the bacteriolytic power of animals which have been immunised to a particular bacterium is dependent on chemical modifications of their blood-plasma, which now contains anti-bacterial substances. These substances, as we shall see, are almost certainly derived from leucocytes, and Buchner and his followers maintain, from their experiments, that they are really of the nature of a leucocytic *secretion*, but the evidence in favour of this view is quite unsatisfactory. Metchnikoff, on the other hand, attributes the presence of these bodies in the plasma or in serum to their *liberation by the destruction of leucocytes*, occasioned by the action of bacteria in the body, or by the process of preparing serum. And such a view regards their origin as similar to that of fibrin ferment in the process of coagulation. The view of Metchnikoff has recently received additional and very strong support from the discovery of Gengou and Bordet, that plasma separated by a special method from the corpuscles without destruction of leucocytes occurring, possesses no bacteriolytic power at all. It follows, therefore, that the leucocytic substance which destroys bacteria is not secreted by these cells into the plasma as a normal process, but only reaches it when it has been set free by a destruction of the leucocytes themselves.

In order to obtain some further evidence, we may now examine what takes place within the bodies of infected animals. Now, when bacteria are introduced into the body of an animal naturally immune to the bacteria in question, they are seen to be rapidly taken up by leucocytes by a process to which Metchnikoff originally gave the name phagocytosis. Cells which possess the property of ingesting foreign particles are spoken of as phagocytes. They include the following groups:—(*a*) All the leucocytes except the coarsely granular varieties, mast-cells, and lymphocytes; (*b*) the phagocytic wandering cells of the tissues; (*c*) endothelial cells, the cells of the spleen pulp, and connective-tissue corpuscles; (*d*) in rare cases nerve-cells (in lepra) and the sarcoplasm of muscle. The first two groups form the free phagocytes, the others being spoken of as "fixed." Their property of taking up bacteria has been already dealt with in our discussion of the inflammatory reaction.

The phenomenon of phagocytic action occurs invariably in animals which are immune or have been immunised, and is a regular event wherever an infection runs a course favourable to the animal infected.

If bacteria be introduced by artificial means into the peritoneal cavities of immune animals it *sometimes* happens that a considerable number are destroyed by extra-cellular action in the peritoneal fluid, and not within the bodies of phagocytes. This fact was first observed by Pfeiffer, in 1894, on greatly attenuated cholera vibriones, and is called Pfeiffer's phenomenon.

Metchnikoff, however, showed that if in this experiment one employs a vibrio of normal virulence, phagocytosis becomes very marked. And this has been confirmed for many other micro-organisms.

It follows, therefore, that the phagocytic action is of great importance in relation to protection. And this was proved quite clearly by the discovery of Sanarelli that anthrax spores, protected from this *direct* cellular attack by being enclosed in a

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collodion capsules developed highly virulent anthrax bacilli within the body of an animal which easily destroyed spores not protected from the phagocytic action.

Metchnikoff, therefore, holds that the essential element in anti-bacterial immunity is phagocytic action, and that the destruction of bacteria is due to the action of bacteriolytic substances contained within the bodies of the phagocytes. He sees the origin of anti-bacterial substances, which may be present in a fresh serum, or in peritoneal effusions, in the extensive dissolution of leucocytes and attendant liberation of their antibodies which occurs when blood is shed, or as a consequence of intra-peritoneal inoculations.

Now, before phagocytosis can occur the phagocytes must first come into contact with the bacteria, either by bodily approaching them, or by extension of their protoplasmic processes, or, on the other hand, by attracting the bacteria to themselves. Accordingly the determining factor of the phagocytic process is to be sought in the phenomenon of chemotaxis. This process I explained in our discussion of the *rôle* of leucocytes in inflammation. And it has been observed that where immunity exists against a given bacterium, that bacterium exerts a positive chemotactic influence upon the leucocytes of the animal in question. This, then, is the visible element which distinguishes immune or immunised animals from those which are highly susceptible to the bacterium concerned. Thus Metchnikoff showed so long ago as 1884 that in unprotected rabbits injected with anthrax bacilli, the micro-organisms multiply rapidly and remain for the most part free, while in a rabbit immunised to anthrax they are quickly ingested and digested by the phagocytes.

In the fact that even in the unprotected animal a few and presumably the less robust bacilli are similarly dealt with by the phagocytes, we find the basis of the immunity which can be artificially induced by the appropriate measures. And this we see is a precisely similar process, as de Barry pointed out, to the habituation of plasmodia, and the accompanying reversal of the negative chemotaxis, to substances normally unfavourable to their metabolic processes.

Hence it appears that anti-bacterial immunity depends on the degree to which the phagocytic process is developed against the particular bacteria in question. And these bacteria are taken up alive in full possession of their normal virulence.

But the serum of an animal which has been immunised against a given bacterium has been proved to be protective for other animals against an infection by the same bacterium. The same we saw was true of antitoxic sera. But while the neutralising action of an antitoxin can and does take place equally well outside the living body as within it, these so-called anti-bacterial or anti-microbic sera, which, on injection into animals, lead to the destruction of bacteria in the body, exhibit no such action on the same bacteria in a test-tube. That is to say, anti-microbic sera have in general no bacteriolytic power—I say in general because, as I shall show you later, they do possess such power in marked degree when absolutely fresh from the body of the animal.

Professor Pfeiffer, starting from this observation that anti-microbic sera possess outside the body no bacteriolytic action, attributed their activity within the body to the intermediation of cells. He found, however, that on the introduction of bacteria such as the cholera vibrio into the peritoneal cavity of an *immunised* guinea-pig, these micro-organisms were destroyed by a process of agglomeration, swelling up, and fragmentation without the intervention of a phagocytic action. The phagocytes remained entirely absent or only began to appear when the extra-cellular process was already well advanced, and such bacteria as were taken up he held to be already dead or dying.

This observation, which we call Pfeiffer's phenomenon, was confirmed and further investigated by numerous pathologists. They found, moreover, that the same phenomenon occurs in unimmunised animals if immune serum—that is, the serum of an animal which has been immunised—be introduced into the peritoneal cavity with the vibriones.

It was next shown that if such immune serum in an inactive condition—that is to say, incapable of killing the bacteria in a test-tube—be injected into the peritoneal cavity of a guinea-pig and samples subsequently removed at intervals, it is found after, say, twenty minutes to have undergone such modification that it has now become actively bacteriolytic, and can destroy the vibrios *in vitro*.

On the results of these and similar experiments Professor Pfeiffer based his theory that the pro-

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tective substances which kill bacteria are modified and active forms of certain antibodies present in the immune serum. The former in their active condition are, according to him, exceedingly unstable substances and easily destroyed. The organism, therefore, stores them in the plasma in a modified and inactive, but highly resistent form, from which it reconverts them when required by means of cellular action. And in the intra-peritoneal experiment this action is ascribed by him to the endothelial lining of the cavity.

Metchnikoff, on the other hand, continued to lay stress on the degree of phagocytosis which he found still to exist in all these cases, and attributed the origin of the protective bodies found in serum entirely to their liberation by destruction of leucocytes. The phenomenon of Pfeiffer is less marked, and phagocytic action the more evident the greater the immunity which the animal possesses, and in highly immune animals bacterial destruction is entirely phagocytic. Moreover, Pfeiffer's phenomenon is more or less peculiar to the peritoneal cavity, which always contains large numbers of leucocytes liable to be rapidly destroyed by an injection; and Mesnil showed that in the subcutaneous tissues the reaction is entirely phagocytic. The same is true of the intra-peritoneal experiment itself after the injection of certain fluids (otherwise indifferent) which produce leucocytosis.

Again, Bordet showed that normal peritoneal fluid can render active an inactive immune serum in a test-tube (Bordet's phenomenon). Here only leucocytes could possibly be present, and there is no question of a modifying action of protective bodies by the endothelium.

Taking the question broadly, it appears that the two great schools of Metchnikoff and Pfeiffer differ not so much on the facts of the phenomena observed, which are in general and with only minor variation fully confirmed by both, as in the construction and meaning which is put upon them.

Both now believe the protective substances to be produced by cellular action, though for the one the cells chiefly concerned are the leucocytes, while for the other the endothelial elements have the greater importance. The endothelial cells, I may remind you, also belong to the phagocytic group of Metchnikoff. Both schools admit the occurrence of phagocytosis, though differing as to its relative importance, and both agree that anti-bacterial sera have no activity against bacteria outside the living body. But, while Pfeiffer holds that its protective action when injected into animals is due to the agency of cells which modify and energise its pre-contained protective bodies, Metchnikoff maintains that this result is rather due to its stimulating action on the phagocytes themselves.

On this latter view the protective action of immune sera is of a similar kind, though specialised and of much higher degree than that which is produced by the injection of such various indifferent substances as I have mentioned under the methods of immunisation.

Whether the breaking up of phagocytic cells, with the resulting liberation of the protective substances which they contain, is simply due to the effect of the injection only, or is occasioned partly by the action of special bacterial products which attack these cells, is not at present certain. But for one bacterium, at any rate (staphylococcus), Van der Welde has proved the formation of a special anti-leucocytic product, which he calls leucocytin. And this discovery affords an explanation of the observation of Muir, that in his experiments large numbers of the leucocytes which had ingested staphylococci and destroyed them, yet subsequently died and degenerated themselves, and were removed by hyaline leucocytes and the large endothelial cells described by Durham.

We saw last lecture that a positive chemotactic influence on the phagocytes is the essential antecedent of their phagocytic action on a given bacterium. And it was proved by Everard, Demoor, and others that as immunity increases in an animal this attraction of its leucocytes by the bacteria steadily increases. Again, it is said that a virulent bacterium may exert a negative chemotaxis, though when attenuated it acts positively. This shows most clearly the relation of virulence to infectiveness, and of the latter to diminished phagocytic action of the leucocytes.

Some pathogenetic micro-organisms practically never succeed in general invasion of the animal body, but only act by means of toxins locally produced. And in such cases it is found that the bacteria in question have a strong positive chemo tactic action on the phagocytes, and hence only

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succeed in gaining *local* foothold in the organism in regions somewhat sheltered from the free access of leucocytes, as, for example, on the surface of the pharyngeal mucosa in diphtheria. Here the animal is to a great extent immune to the bacterium, although highly susceptible to the action of its toxic products; that is, it possesses anti-bacterial immunity, not antitoxic.

The same distinction holds experimentally, for animals immunised to a bacterium often remain susceptible to its extra-cellular toxins. This is to be explained as follows:—The amount of toxins introduced in each inoculation of the bacterial culture is quite small, and insufficient to induce the formation in the body of an antitoxin. Meanwhile the phagocytes acquire the power of rapidly destroying the bacteria injected, and thus prevent them forming further toxins in the animal body. This phagocytic power increases as the immunity progresses, and the bacteria are more and more rapidly destroyed at each injection. Accordingly they never have an opportunity of forming toxins in the animal, whose serum therefore gains no antitoxic action.

That the explanation just suggested is the true one follows from the fact that if such animals be subsequently treated with the toxins they are found to yield an antitoxic serum, and to now possess not only bacterial but also toxin immunity.

Now, on the injection of bacteria in a relatively immune animal there occurs not only chemotaxis of the existing leucocytes, but also rapid increase in the leucocyte formation, resulting in the production of leucocytosis. This occurs also in the course of the acquisition of immunity during immunisation. The process has its seat in the bone-marrow, and is the result of stimulation of the marrow-cells to an increased proliferation by the chemotactic action of bacterial products, as was shown by Ehrlich, Goldscheider and Jacob, Muir, and others.

The visible element, then, in a bacterial immunity is the ingestion of the infective agent by the phagocytes and the increase in number of the phagocytic cells to meet bacterial invasion. Now these bacteria are taken up alive and fully virulent. This has been proved by Sanarelli and Cantacuzène, who further showed that if the vitality of the phagocytes be lowered in any way—as by the action of cold or the administration of opium to the animal—the ingested micro-organisms at once proceed to multiply within the body of the phagocytes, which they kill, and, thus becoming free, lead to the death of the animal which was previously immune.

We therefore need to seek an explanation of how the bacteria are killed in immune animals after their ingestion by the phagocytes. Here we require to turn from the consideration of the biological to that of the more evidently chemical factors in bacterial immunity. This is an exceedingly difficult and complicated question, though it has been greatly simplified by the discovery and preparation of the so-called anti-cellular sera, which are homologous in their action with the anti-bacterial sera, but are for several reasons easier to investigate.

It has long been known that the blood of an animal undergoes rapid and complete destruction when injected into the circulation of an animal of a different species. On the other hand, it had been discovered that the blood of certain animals is poisonous for other species when injected; as, for example, that of eels, of snakes, of toads, and of the hedgehog. And Calmette showed that animals can be immunised against such toxic action just as against the toxic products of bacteria.

Belfanti and Carbone then arrived at the important discovery that the blood of horses, which has no toxic action when injected into rabbits, can be made toxic for these animals by the repeated injections of rabbit's red blood-corpuscles into the horse. The activity thus acquired by horses' blood was found to be specific in its nature; that is to say, the blood was only poisonous for rabbits, not for other animals. This property was shown by Bordet to depend on processes analogous to those occurring in the formation of anti-microbic sera. And similar specific changes have been demonstrated following the injection of various other elementary cells; for instance, different kinds of epithelia, leucocytes, spermatozoa, and kidney cells, and so on. The sera thus produced are spoken of as anti-cellular sera.

The anti-sera for red blood-corpuscles are called *hæmolytic* sera. If a fresh specimen of such a serum be allowed to act upon the red corpuscles of the animal for which it is specific, it rapidly produces their solution. This is occasioned by the action of *two* substances which it contains, and which were first identified by Bordet.

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Pfeiffer had already stated that anti-microbic sera similarly contain *two* bodies necessary to bacteriolytic action. To these the name of immune body and of addiment or complement were respectively applied by Ehrlich.

Fränkel and Soberheim had further shown that if an actively bacteriolytic anti-cholera serum be heated for a time at 60° C. it loses its bacteriolytic power entirely; and Bordet found that its activity can be restored by the addition of fresh normal serum which by itself had no bacteriolytic action. Bordet accordingly came to the conclusion that one of the substances in question, namely, the complement, is present also in fresh normal serum as well as in specific immune serum, but that it is readily destroyed by moderate heat; and that the other substance—immune body—which is more resistent, is not, in general, present in the serum, but is a specific product formed during the process of immunisation. He established the same facts for hæmolytic sera.

Buchner had already found that hæmolytic action is destroyed at a temperature of 56°. Bordet now gave the explanation of this fact by showing that the immune body, which is special to the specific serum, is not injuriously affected by such temperatures, but that the complement, a body present also in the sera of unimmunised animals, is more unstable, and is rapidly destroyed by heat.

These observations attracted much attention, and gave rise to numerous researches by Ehrlich and Morgenroth, Metchnikoff, Bordet himself, and many others. The experiments carried out by these investigators, and more especially by the two first named, have resulted in the addition of the following facts to our knowledge of the composition and the mode of action of these sera:

1. The immune body and the complement exist side by side, but ununited, in the immune serum.

2. The immune body, which is specific, has a strong affinity for the red corpuscles, and rapidly unites itself with them even at a temperature as low as 0° C. It may thus be separated from the complement, which at this temperature remains unattached, by means of centrifugalisation and washing of the corpuscles.

3. The complement is only taken up by the corpuscles at a higher temperature, and only then if the immune body is also present. In the absence of this body it is never taken up at all by the red cells.

4. Neither of these bodies alone can cause solution of the red corpuscles; both must be present and be taken up before a hæmolytic action is produced.

Hence Ehrlich came to the conclusion that since the immune body is readily taken up, but of itself produces no effect in the absence of the complement, while the latter by itself produces no hæmolysis, and is not even taken up by the red cells, the process of hæmolysis, when occurring, must be due to an action of the complement through the agency of immune body.

Bordet put forward a somewhat different explanation of the facts observed. He holds that the immune body so changes the red corpuscles that these themselves now bind the complement, which thus causes their solution; that is to say, the complement becomes attached not through an intermediate link of immune body, but independently.

Ehrlich, however, showed that there appears to be a direct connection between the two substances which cause hæmolysis; since, if the complement of an immune serum be destroyed by heat, leaving the immune body intact and isolated, the former can be readily restored by the addition of serum from a normal (unimmunised) animal of the same species, but is not *necessarily* replaced on the addition of serum from another species.

An explanation of these observations has been offered, based on an extension of the side-chain theory which I previously described.

On this hypothesis the immune body is a lateral chain or cell-receptor, which is produced and separated into the blood in the same manner as is an antitoxin or the other anti-substances. It is supposed, however, to have two haptophores, instead of only one, as was the case with antitoxins. One of these haptophores is endowed with high affinity for the red corpuscles, of which it constitutes the specific antibody, while the other possesses a similar affinity —though somewhat less since it does not anchor the complement at low temperatures—for the complement.

It was next shown by Von Dungern that the specific immune body and the complement are not present in equivalent masses in an immune serum. Thus he succeeded in increasing the hæmolytic action of his rabbit's sera more than thirtyfold by

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simply adding complement in the inactive fresh serum of a normal rabbit. In other words, the hæmolytic serum here contained no less than thirty times more immune body than complement. And hence it follows that the two bodies are not produced in combination and so set free into the plasma, since in that case they should appear in serum in equivalent amounts. And Dungern came to the conclusion that while the immune body is a product of immunisation, the normal complement is not increased at all during this process. The fallacy of this conclusion I shall show you later.

Ehrlich and Morgenroth have proved that the processes at work in the reaction take place quantitatively; that is to say, that the red corpuscles employed in the experiment take up just so much immune body as is needed for their complete solution and no more. They further showed that the ferment-like working complement is not in every case the same for all the hæmolysins which can be produced in the same animal or the same species, since they found sometimes in the same animal, besides the thermo-labile complement which is destroyed at 56° C., a more resistent thermo-stabile form which can withstand this temperature for several hours.

Buchner and his pupils had already investigated the hæmolytic properties of *normal* sera; these also are destroyed by moderate heat, and exhibit in other respects conditions closely akin to those existing in the artificial hæmolysins. For these sera also Ehrlich proved the presence of an intermediary body analogous to the immune body found in the serum of an animal which has been specifically immunised.

As regards the complement, Ehrlich and Morgenroth found that this may be replaced in a large number of instances, though not in all, by the fresh normal serum of an animal of a different species. It therefore is not a specific body, but a normal blood constituent. Into the question of its origin and nature we shall enter later on. Meanwhile we must examine next the conditions governing the formation of the immune body.

Now, on the side-chain theory of Professor Ehrlich, the only necessary condition for the formation of an antibody to any substance which may be injected is the existence in some group of cells within the body of suitable side chains or cell-receptors which can take it up. And such receptors when discharged into the plasma are actually the antibody in question.

Considering, then, the great number of the organs and tissues of the body and the manifold variety of their metabolism, such side chains, which by supposition are the normal agents in the nutritive exchanges, must exist in the most extraordinary diversity in the body-cells, and also on occasion in the blood itself. And hence the blood must undergo in respect of the side chains discharged into its plasma both constant and considerable variations. This is proved, moreover, by the observations that a normal horse's blood may at one time contain a considerable amount of anti-rennin, at another none, and that dog's blood may be at one time highly hæmolytic for blood of cats, while at another it exhibits no such hæmolytic action.

As regards any substance injected into a living animal, three possibilities alone present themselves. First, it may find no suitable receptor in the body-cells; secondly, suitable receptors may be present; or, thirdly, with the presence of suitable receptors there may also be present in the body in other cells a group possessing the same haptophore as the injected substance. For simplicity let us call the chemical group which is injected the male receptor, and the group which takes it up within the body the female receptor. Then the three possible cases are as follows:—Either (1) the animal contains no female receptor; or (2) it does contain the female receptor, and this alone; or (3) it contains both the female and the male receptors.

And this third case, in which there is the simultaneous presence in the animal of corresponding male and female groups, is probably of very frequent occurrence in the economy of the animal, and has to do with the dependence of certain cells for nourishment on the products of certain other cells within the body. This occurrence, for example, might explain the relation of the testicle to the prostate, and of the ovary to the mamma, so familiar clinically, but so little understood. It is exemplified in the proved co-existence of the rennet and the anti-rennet groups in the same animal.

Such receptors may be called "reciprocal," the term "singular" receptor being used in cases where the corresponding or male group is not already present

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in the animal. The three cases, therefore, may be given as follows:—(1) No receptor present; (2) female receptor present only—singular receptor; (3) reciprocal receptors present—both male and female groups existing in the body of the animal. In the first case, evidently, no antibody can be formed, since there is no receptor to take up the injected substance. In the second case an antibody will be formed, and there is no particular condition limiting its quantity; this is exemplified in the formation of antitoxins. In the third case an antibody is also formed, but this on its appearance in the plasma will be taken up by the reciprocal receptor, and consequently will never be found free in very large amount in the blood-plasma. And this was actually shown to be the case for anti-rennin.

These possible cases have been carefully worked out by Ehrlich and Morgenroth for hæmolysins, but I have only time to give you a brief summary of their results, which were obtained on goats.

If a goat be injected with the blood of other goats it forms a hæmolysin for their red corpuscles. This may be called an isolysin to distinguish it from lysins formed for the red blood-corpuscles of animals of a different species from the one injected, which are called heterolysins. A goat can thus form isolysin for the blood of each and every other goat whose blood is suitably injected. The isolysins formed are not identical, but different, and none of them is hæmolytic for the red corpuscles of the goat which forms them; that is to say, the animal will not form hæmolysin for its own red corpuscles. The isolysin formed is therefore not an autolysin for the blood-cells of the animal concerned. Indeed, no formation of an autolysin has ever been observed, and from this it follows that animals do not contain receptors which could aid in the destruction of their own tissue-cells. Again, the injection in an animal of the hæmolysin of its own red corpuscles leads to the formation of another antibody, an antilysin; and hence it follows that even should an animal possess the power of forming lysin for its own cells, this lysin would be quickly neutralised by the production of the corresponding antilysin.

As regards other anti-cellular sera, the facts, so far as they have been established hitherto, fully confirm these observations on the hæmolysins.

The experiments throw a valuable light on the formation of antibodies in general and of antitoxins in particular, since they show that wherever susceptibility to the action of an injected body exists, owing to the presence of suitable receptors in the cells on which that body exercises its specific action, there exists also of necessity the basis of immunity in the power of splitting off those receptors from the cells in question and discharging them into the plasma as an antibody.

In the absence of such receptors from these cells, whether they be present or not in other cells of the body, the animal will evidently exhibit a natural immunity to the specific action of the toxic substances injected, because the latter is not taken up from the blood-plasma by the cells in question. Thus the toxin of tetanus, which acts especially on cells of the central nervous system and is taken up by those in a susceptible animal, is found to be totally absent from the nervous tissues after its injection into animals naturally immune.

If the naturally immune animal possess a suitable receptor for the toxin in other cells than those for which it is specifically toxic, these will remove it from the blood and store it up, as is the case for tetanus toxin and the liver-cells of scorpions, which are naturally immune to tetanus.

And such receptors will produce an antitoxin, although the animal is naturally immune. Thus Metchnikoff showed that crocodiles, which are immune to tetanus, but whose blood normally contains no anti-tetanus toxin, produce an antitoxin of considerable value after injection with the tetanus toxin.

Since any given antibody simply represents the female cell-receptor of the injected substance which has been discharged into the plasma of the animal concerned, it follows that all substances whatever which are endowed with the *same* haptophore, and therefore have an affinity for the *same* receptor, will on injection lead to the formation of the *same* antibody. A given antibody, therefore, will be protective against all toxic substances which possess a common haptophore. This fact suggests a simple explanation of the observations which I previously mentioned, in which inoculation with one micro-organism or its toxins was found to be protective also against certain others.

The immune bodies formed against bacteria have the same relations as any other of the anti-substances. So far as evidence goes, they are produced under precisely similar conditions.

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CLINICAL LECTURES

ON

DISEASES OF CHILDREN.

LECTURE I.

THE CLINICAL EXAMINATION OF SICK CHILDREN.

Delivered at the London Hospital, November 1st, 1902.

By ROBERT HUTCHISON, M.D.Ed., M.R.C.P.,

Assistant Physician to the Hospital, and to the Hospital for Sick Children, Great Ormond Street, W.C.

GENTLEMEN,—It will be generally admitted, I think, that the study of diseases of children, which will occupy us in these lectures, is one which is apt to be neglected in general hospitals. One reason for this probably is that adult patients are so numerous, and their claims on the staff are so pressing, that there is very little time left for special teaching in the children's wards. And also, I think it has its explanation in this further fact, that the teaching of general hospitals is, and necessarily must be, directed specially to the requirements of qualifying examinations. Now it is, in my judgment, at least, unfortunate, though I can hardly expect to carry your sympathies in this matter, that there is no demand made at the ordinary qualifying examinations for a special knowledge of the diseases of children, the consequence of which is that many men when they qualify know almost nothing of many of the commonest ailments of infant life. You go out into practice and you are not much better informed in many cases than the mother or the nurse as to the diseases which you have got to deal with. And yet you will find that in any large general practice children make up a great part, perhaps the greater part, of your patients, and the seriousness of their diseases will be brought home to you by a study of the death-rate, for whereas the adult mortality has gone down in the last fifty years by about four per thousand,

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the infantile mortality is as high as ever it was; and, indeed, of every hundred children born fifteen never see their first birthday. Now, gentlemen, that is not creditable to our profession, and although I admit that much of it is due to social and economic causes for which you and I are not in any way directly responsible, yet I cannot doubt that if all practitioners had the opportunity as students of becoming familiar with the treatment of the common diseases of infancy, the result *would* be seen in an appreciable diminution of our infant mortality.

I should be sorry, however, if you were to embark upon this course of study merely from such utilitarian motives as those I have put forward, because I can promise you that on scientific grounds alone, alike pathological and therapeutic, there is no subject which will better repay your attention than the study of the diseases of children. You will find that pathological processes in children tend, if I may use the term, to be pure bred; they are not hybrids produced by a blending of different morbid factors. Disease in children also runs a less complicated course than it does in the adult, and tends to exhibit pathological processes in their simplest forms.

As regards therapeutics, you will find treatment in children eminently satisfactory. I am bound to confess that when you study the results of the treatment of disease in adults in large hospitals such as this, you are tempted to form a rather low opinion of the potentialities of medical therapeutics. Frequent failure is inevitable; it depends largely on the circumstances in which our patients in this part of London are placed. But with children it is not so; they respond to treatment in a most surprising and gratifying fashion. One reason is, no doubt, that you can so easily modify your patient's surroundings. A child has not got to work; you can take him away from school for an indefinite number of weeks, and no one will object except the schoolmaster or the attendance officer. Moreover, in the case of children you are fighting with Nature instead of against her. In the case of most of our adult patients they are going down the hill, or are about to go down it; but children are going up the hill, and they have great reparative power to fall back upon, and hence it is that the treatment of their diseases is so encouraging. All the more reason, therefore, that you should know how to treat these diseases, for you will often find when you go into practice that the life of the child is literally in your hands, and that a mistake at a critical moment may be really a matter of life and death.

Now, it has always been the boast of this hospital that whatever it may do or leave undone, at all events it offers students unrivalled opportunity of fitting themselves for practice. But although this is true, and although we treat here a much larger number of sick children than any other general hospital, there has not hitherto been given here, any more than at any of the other Metropolitan Schools, systematic instruction in the special diseases of childhood. That gap in your training I should consider it a privilege to be able to fill, and I have therefore invited you to meet me here, by the consent and with the encouragement of my colleagues, in order that we may consider together, briefly and practically, the nature and treatment of those diseases which are peculiar to early life.

I may now turn, gentlemen, to the subject of the first lecture of this course—the methods of examining children clinically. I must ask you to bear with me if you find this not extremely interesting, but it is an unavoidable preliminary, for the methods of examining and handling a sick child do not come to you by intuition. It was pointed out by Dr. Charles West, who was one of the pioneers in this subject, that the man who starts for the first time to study the diseases of children is like a traveller in a foreign country; he hears a strange language spoken which he does not understand. At all events, if the language is not absolutely strange, it is spoken with a foreign accent, for the physical signs of disease are often different in children to what they are in grown-up people, and so you have to devote special attention to their interpretation.

Begin with the history. The child, of course, will be unable to tell you anything, or but very little, and you have to fall back upon the information supplied by the mother or the nurse for a history of the illness. These are apt to be long-winded persons, but it is always worth while to listen patiently to all they have to say, for there is nobody in such a favourable position to observe small changes in health of the child as those who are constantly attending to it. Listen attentively,

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then, to what the mother or the nurse has to say as to the mode of onset of the illness, and the symptoms which the child exhibits.

With what are called "subjective symptoms" in children you are not in any way troubled, because there are none. Pediatrics is like veterinary work in this, that the patient is unable to give you an account of his sufferings, and you are thrown back entirely on your own observation, which, of course, makes the necessity for careful examination all the greater. With regard to the history, I would also say that nutrition and heredity play such a large part in the diseases of children that you must always make a special inquiry into these points. You must inquire, as regards the family history, what other children there have been, how many of those are living, and how many, if any, are dead, and the ages at which they died; and you must project yourself back into the intra-uterine life of the child as far as you can, and inquire about the health of the mother during pregnancy, and whether the child was carried to term or not. You will also inquire as to the nature of the labour by which the child was ushered into the world. All these points have a direct bearing on the conclusions which you will draw as to the kind of heredity with which your patient is burdened. You will further inquire with regard to nutrition, noting the exact mode in which the child has been fed, as well as the particular food or foods which are being given now.

Answers to these questions will clear the ground, and will prepare you for proceeding to your physical examination. I need hardly emphasize here the importance of gentleness and tact. These things cannot be taught; they come naturally to some people; but everybody can acquire some degree of them by care and with opportunities for practice. Children are difficult to handle; they are easily frightened, and they distrust the presence of a stranger, and these things add to the difficulties which you will encounter. The methods which you adopt in your examination of children are the usual methods of inspection, palpation, percussion, and auscultation. These have not the same relative values, however, as in the case of adults. Inspection is immeasurably the most important method in a child. The next in importance is palpation, next auscultation; percussion we will leave till last.

You have already, of course, gleaned much information by casual observation whilst you have been talking to the mother and getting the history. During that time the child has become accustomed to your presence. You will have noticed whether he looks seriously ill, whether he is interested in his toys or surroundings, and whether he presents any obvious signs of disease. Having done that, you have to proceed to more exact inspection. For that purpose it is important to have the child completely stripped. It is well that this should be done before you come into the room; it is bad to get a baby to associate you in its mind with the taking off of its clothes. The next piece of advice I would give you is never to look a baby in the face. That, of course, does not apply to grown-up persons; we feel inclined to distrust a man who does not look us in the face; but the baby distrusts the man who does. There is no surer way of making a baby cry, and therefore of making it far more difficult to examine, than by staring it in the face.

You should begin your inspection by noting the child's expression. Much has been written about the facies of disease, and you will find mysterious lines described as coming out on the child's face when it is ill. I do not think any good purpose would be served if I attempted to describe them to you. That is a matter which you must pick up as you have opportunity here, where you will be able to see the facies of all sorts of diseases in infants, and you will get familar by-and-by with the facies of abdominal disease, with the facies of acute respiratory disease, of cerebral disease, and so on. The importance of this subject, however, justifies all that has been written about it, because the face of the child is a mirror in a way in which that of a grown-up person is not. It is a clean sheet upon which disease can write; it is not furrowed over with the lines of care and anxiety, nor does it show those changes so often to be seen in adults as a result of debauchery and bad living. Moreover, the signs of disease in a child are not masked by the emotions nor suppressed by the will.

Now, having noted these things, you cast your eye down over the child, and you will observe certain peculiarities of configuration which are normal, but which the child's mother may think are abnormal. One of these is that a child has a relatively large abdomen. You will not be long in practice before

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babies are brought to you by anxious mothers with the complaint that "the stomach is getting large." I do not say that these mothers are not occasionally right, and that the abdomen may not be pathologically enlarged sometimes, but it is well to remark that a child's abdomen is naturally prominent, the main reason for which is that the liver is relatively bigger and occupies a considerable space in the abdomen.

Another peculiarity is that the chest is very round, and it is only after some time that it acquires the oval shape which is seen in a well-formed adult. There are certain consequences of that round-shaped chest in children which I shall have to point out to you when we come to study respiratory disease. Another point to note is the relatively large head of the child compared with the trunk. Here, again, anxious mothers come to you complaining that the child's head is getting large, and possibly the neighbours have been trying to make her believe that the baby has got "water on the brain." Remember in such an event that a child is about three years of age before the head and chest have the same relative size as in the adult. Of course this is easily understood when you remember that during those years the child's brain is growing rapidly.

Other points to notice are the character of the respiratory movements; whether they are rapid, regular, easy, or laboured. You will also notice whether there is any head retraction. You will observe the position of the limbs; whether they are held in an easy and natural posture, or kept rigid, as they are apt to be when the seat of painful disease such as scurvy. Look also for the signs of rickets, which you will quickly learn to recognise when we come to speak of that disease. You will, at the same time, notice the presence or absence of rashes, which are so important a diagnostic sign in the diseases of infancy.

Having run your eye rapidly over the child in the way I have mentioned, we come to the next method of examination, namely, *palpation*. The advice used to be given by an old teacher of mine that you should always "paw your babies." That was very sound advice. But I would remind you of the importance of a warm hand in doing so. Though it is now far back in point of time, I remember still having been handled by a doctor with very cold hands, and I never forgot it or forgave him. I recommend you to begin palpation with the head. You do not often palpate the head of a grown-up patient, but in the case of a child it is very important to do so. First of all go over the anterior fontanelle; notice whether it is closed or open, bulging or depressed. The fontanelle tells you as much in a baby as the pulse does in older patients, a depressed fontanelle having the same significance as a feeble pulse. Notice whether there is any sign of softening of the bone, such as one finds in cranio-tabes, which is a frequent sign of rickets, or syphilis. Observe also whether there is any bossing of the head, and whether there are any nodes or lumps upon the bones.

Having studied the head, you can proceed to the trunk, passing your hand over the skin, and noticing whether it is hot or cold, dry or moist, for the latter is information which no thermometer can ever give you. You will also notice whether there is beading of the ribs, which is an important and early sign of rickets; then feel for the edge of the liver. You can generally make it out quite well a little below the costal margin. The spleen, of course, you ought not to be able to feel in health. Lastly, I think you should run your hands over the limbs and notice whether there is any tenderness of the legs. Many cases of obscure disease in children are to be explained by inflammatory affections of the bones, which can only be determined by careful palpation.

We pass next to *auscultation*. One should perform this before percussion in children, reversing the rule which obtains in the case of older patients. Percussion is apt to frighten children, and so would make auscultation difficult if that were left to the last. In auscultating the chest it is best to have the child sitting up; it should not be lying on its face, for the child is likely to have unpleasant associations with that position, and also because the pressure on the abdomen, if the child is on its face, pushes up the abdominal organs and interferes with the expansion of the lungs. Therefore examine the child when it is sitting up and leaning against the mother's or the nurse's shoulder. It is commonly recommended that you should listen to the chests of children without a stethoscope, by putting your ear to the back. Certainly by that means you frighten the child less than by any other method, but it is open to objection in patients who are not very clean. If you use a stethoscope it

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must be a binaural. You have to follow the movements of the child as it wriggles about, and you cannot do that with a single stethoscope. Further, do not have a stethoscope with a metal end-piece; have vulcanite, or something which is warm when put upon the skin. It is an advantage to have a short chest-piece, so that you can get in between the mother and the child, and get round corners without interfering with the child's position. It is of help to have a reversible chest-piece with a narrow end to go into the small spaces between the ribs. You may think that the crying of the child is a great bar to auscultation, and you will find it so as a beginner, but by-and-by when you get practice you will rather prefer that the child should cry a little, because crying ensures full expansion of the lungs, and is the equivalent in the adult to the taking of a deep breath.

You will be struck at first by the length of time that a child can hold its breath, and you may have to wait for quite a long time for another inspiration. This is not a sign of disease; it is merely due to the enormous relative vital capacity of the infant chest. There is another peculiarity of respiration met with in children who are the subjects of serious respiratory disease, which is likely to confuse you at first, and which is best described as an "*inversion of the respiratory rhythm.*" You and I, when we breathe, first fill the chest, then empty it, and then stop for a moment; that is to say, the ordinary rhythm is inspiration, expiration, pause. But you will often notice in children in whom respiration is embarrassed that a short grunting expiration is made, followed by a full inspiration, and that by a pause. In other words, the ordinary rhythm has become inverted. What the advantage of this mode of breathing is it would be hard to say. It has been suggested to me by my friend Dr. Sutherland, who has given some attention to the matter, that the result of such breathing must be to facilitate free oxidation of the blood, for the pause after inspiration means that the lungs are kept full of pure air for a much longer period than by the normal method. Such a reversal of the usual mode may be possible to the child by reason of the greater plasticity of its respiratory apparatus and by the fact that the mechanism of respiration is not so stereotyped as it becomes in the adult. At all events, whatever the explanation may be, you will find the occurrence of this inversion of respiration of considerable diagnostic value in many cases.

There are other points in connection with auscultation of the lungs in children which have to be noticed, and one of these is the harshness of the respiratory sound. This is the so-called puerile breathing, and it is so marked and constant that, if you hear loud breathing on one side of the chest and faint on the other, the loud is probably the normal. Towards the right apex, and between the scapulæ behind, the respiration in the young child is normally not only harsh, but almost bronchial, a point which it is well to be familiar with if one is to avoid errors of diagnosis.

Another noticeable thing is the extreme ease with which sounds are conducted in a child's chest, so that if you hear a faint accompaniment on one side it is possible that it is really being produced on the other. In the chests of children, too, you meet with pathological conditions which you do not encounter in grown-up persons; for example, collapse of the lung. It is one of the first things to think of in considering the abnormal sounds in a child's chest, whereas it is one of the last things to consider in the case of an adult.

Passing to the auscultation of the heart, I would remind you that the blood pressure in the child is low. In a young infant the capacity of the heart is to the cross diameter of the arteries as 25 is to 20. At the age of puberty the proportion is as 290 is to 61. This means that there is a relatively large channel for the blood to pass into in the child, and that there is no great obstacle opposed to the heart; hence the low blood pressure. In consequence of this the first sound is louder than the second in all the areas, for owing to the low blood pressure the second aortic sound is comparatively feeble.

There is also a difference in the child between the relative loudness of the pulmonary and aortic second sounds at the base. In the adult the aortic second sound is accounted louder than the pulmonary. In the infant that is not the case. Below the age of four—and this is based upon the investigation of one thousand cases*—the pulmonary second sound is decidedly louder than the aortic. From twenty to forty careful observations show that the sounds are about equal. Above forty the

* See a paper by Miss Sarah Creighton in the 'Medical Record,' January 13th, 1900.

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aortic second sound becomes progressively louder than the pulmonary. This means that if you are listening to the heart in a young child you are to regard as accentuation of the aortic second sound anything which is equal to the pulmonary.

You will also note that the rhythm of the heart in little children is often irregular, and you must not attach any importance to that, particularly if the child is asleep. Note also the comparative frequency of congenital bruits. If you hear a loud murmur below the age of two it is almost certainly congenital, because endocarditis hardly ever occurs at that age. Hæmic murmurs, however, may occur below two years. This has been denied by some writers, but there can be no doubt of the fact, and such murmurs have been mistaken for the results of organic disease.

The study of the pulse is of little importance in the case of young infants. If you want to count the rate of the heart you do it by listening with the stethoscope, and if you want to learn anything about the tension of the blood you do it by observing the fontanelle.

Lastly, we come to *percussion*, and here let me say at once that it is not necessary to use a heavy stroke. Do not percuss the chests of little children forcibly. You learn more by lighter percussion, and you do not give the child the impression that you are punishing it. It is often an advantage to use three fingers, for by that means you throw a broad area into vibration. I ask you to remember also that you may get certain sounds normally in infants which are abnormal in old people, such, for example, as the cracked-pot sound. You can often hear a distinct cracked-pot sound on percussing the apices of a crying child as the air is driven out from the apex of the lung through the mouth. That is due to the great elasticity of the chest-wall of the child, which yields before the percussing finger. Be careful also when you are percussing the chest of a child to percuss during the same phase of respiration when you are comparing the two sides of the chest. You will find that that makes far more difference in the case of children than it does in grown-up people, and I have known forgetfulness of this fact lead to the belief that there was dullness, whereas one was only percussing one lung during inspiration and the other during expiration. Sometimes you will detect dulness over the manubrium of the sternum in a child, and you must remember in such a case the possibility of an hypertrophied thymus or enlarged bronchial glands.

Having carried out these methods of investigation, you will look lastly at the tongue and throat. The importance of examination of the throat of the child I cannot exaggerate, because so many diseases of children are associated with abnormal conditions of the fauces. Here you may have to use some force in order to open the child's mouth. There are many ways of coaxing a child to open its mouth, but you may not have any time to waste over them, and may have to proceed to more energetic measures. One of the best ways is to push the lower lip over the lower incisor teeth, and then to press downwards; in order to take the lip away from the teeth the child will open its mouth. Sometimes you may require to hold the nose till a gasping breath is taken. You examine the throat in the usual way, taking care that you get plenty of light, and in many cases you may also require to proceed to palpation of the throat, but that I shall describe when speaking of the part played by adenoids in the diseases of children. The examination of the nervous system and special senses I shall not describe here.

Finally, gentlemen, you must not expect to be very systematic in examining children. I have spoken as if you proceed systematically, but when you come to investigate a case you have to seize the opportunity of examining an organ when it presents itself. When the child is on its back you must examine the heart, the anterior aspects of the lungs, and the abdomen, and when it is sitting up you have to examine the back of the lungs and the spine. You have to run over everything quickly as opportunity offers.

There are still one or two special points to be mentioned. The first is the *weight and height* of the child. I cannot impress upon you too strongly the importance of weighing babies. In the children's hospital I have every baby weighed before I see it at all. The weight is often the only criterion as to whether the infant is improving or going back in health. Here I show you a simple weighing apparatus which has been introduced. The child is laid in the basket and the weight is read off on the scale. I do not mind what weighing apparatus you use, but you must weigh all your children so far as you can. I have put into your

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hands a table showing what weight a child should be at different ages, and I have here a chart showing the curve of weight for the first year. There are certain turning-points of weight to which I would direct your special attention, and which I hope you will be able to remember. At birth the average weight is 7 lbs. At five months old the weight is 14 lbs., at eighteen months 21 lbs., at six years 42 lbs., at fourteen years 84 lbs. These weights, being multiples, can be easily kept in mind.

Another important point is the *circumference of the head.* You should remember that the normal is thirteen inches at birth; at the ninth month it is seventeen inches; at one year it is eighteen inches; at five years it has only grown another two inches. This will show you the enormous rate at which the child's head grows during the first year of life, growth which is due to the rapid enlargement of the brain.

In conclusion, there are certain *milestones*, as I may call them, in the development of the child, to which I should like to draw your attention, because they are points with which mothers and nurses are familiar, and which you will look foolish about if you do not know them. The first is the time or age at which the child *cuts its teeth.* This begins at the sixth month. Of course you will look absurd if you ask whether a three-months-old baby has got any teeth. All the temporary teeth are present at the age of three years. The permanent teeth begin to come at the age of six years instead of six months, and by the age of twelve all except the wisdom teeth are present. The second milestone is the *closure of the anterior fontanelle*, which occurs between the eighteenth and twenty-fourth months. If it is not closed by the twenty-fourth month there is something wrong. The third set of milestones is associated with the *assumption of the erect position.* A healthy baby will hold up its head at three to four months; before this it merely waggles its head about as if trying to balance it on the neck. If the child does not hold its head up by the age I have mentioned, you should suspect some impairment of mental development. The infant sits up at from nine to twelve months, and will walk at from twelve to eighteen months. It should talk well when it is two years old.

The last thing I want to mention to-day is the *character of the motions* in a child. Alterations in the fæcal evacuations constitute another set of milestones in development. Remember that for the first eight weeks there should be three to four motions daily. You are not to regard this number of motions at such an age as evidence of diarrhœa. Up to this period the motions resemble beaten-up eggs in colour and consistence, and they are of somewhat sour but not feculent odour. Look specially for cheesy masses of casein in them, for if you find these you know that the child is getting more milk than it can properly digest. Up to the end of two years from about the eighth month there is an average of two motions daily. They become now more brown in colour, more porridgy in consistence, and of a slightly feculent odour. After two years of age the motions are well formed and feculent.

In my next lecture I shall deal with the subject of infant feeding.

THREE LECTURES
ON
COMPLICATED FRACTURES AND THEIR TREATMENT.

Delivered at the Medical Graduates' College and Polyclinic, March, 1902.

By C. B. KEETLEY, F.R.C.S.,
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LECTURE III.

FRACTURES INTO JOINTS.

Diagnosis.—We require, for diagnostic purposes, a fair but not minute knowledge of the anatomy of the joint injured, especially as regards the normal relations of its salient points to one another, *e. g.* in the case of the elbow, of the relative positions of the two condyles, the olecranon, and the head of the radius.

We should compare together carefully the two corresponding joints, the left and the right. Again taking the elbow as an example, the plan I follow is to cause the patient to place his two hands one on the other on the top of his head, and then to bring the elbows as near together as can be managed in front of his face (*vide* Fig. 3).

There are then two triangles to be compared. The base of each is formed by a line uniting the tips of the two condyles. The apex is the end of the olecranon (*vide* Fig. 4).

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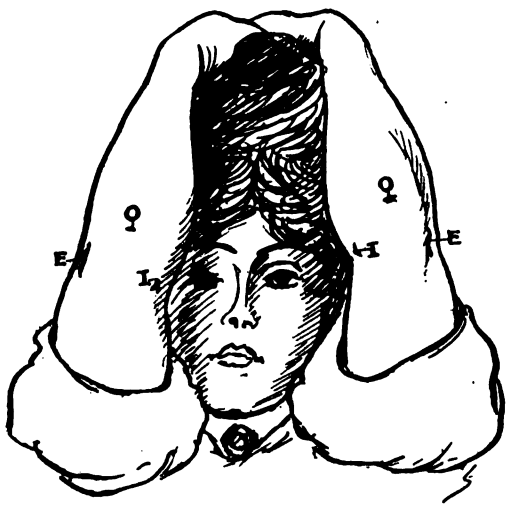
In a diagram of this triangle let O represent that angle of the olecranon which forms the point of the elbow, let I be the internal and E the external condyle. Any general effusion into the joint will cause a puffiness between O and I. If the effusion is considerable it will extend between E and I. If there is a fracture between the condyles into the joint there will be increased distance between E and I, best measured by a pair of calipers.

If the olecranon is broken off its tip will descend

Fig. 3.



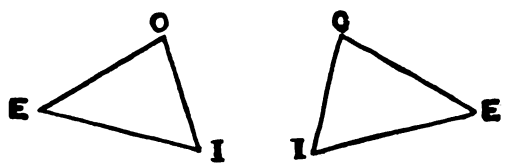


Fig. 4.

from O towards the line E I. The end of the lower fragment, *i. e.* the broken end of the main part of the ulna, must not be mistaken for the point of the elbow. Any doubt on the point can be removed, not only by discovering the "upper fragment," but also by comparative measurements taken from the styloid processes of the two ulnas.

Fracture of either condyle, with displacement, disturbs the relation of E or of I to the other two points. The head of the radius should, from this point of view, be more or less hidden by the condyle E; but if the radius is dislocated outwards or backwards, its head will become more prominent than the adjacent condyle.

If there is a supra-condylar fracture or a complete separation of the lower epiphysis of the humerus, it will probably be discovered either before the elbow is raised to the position I am recommending, or, unless the patient is anæsthetised, during the act of raising the elbow to the position; because a complete transverse solution of continuity of the humerus generally makes raising the elbow on high a painful procedure. In this case the two elbow triangles may be compared by placing the upper arms close to the flanks, flexing the elbows to a right angle, and then viewing the patient *from behind.*

Almost the only elbow fractures in which this method gives merely negative information are those of the coronoid process of the ulna and of the head and neck of the radius.

Great swelling may interfere with the use of this method of ocular comparison; but great swelling, and still more great ecchymosis, should cause a strong suspicion of fracture, unless limited mobility under anæsthesia points to dislocation. Remember that dislocation may co-exist with fracture, *i. e.* with increased mobility of the broken part. I am, of course, leaving out of account the limitation of movement by pain and tenderness.

The head and neck of the radius should be examined by direct palpation, while the forearm is being pronated and supinated.

A note being made of the information got in this way, the next step is to submit doubtful cases to the X rays. Both a front and a side shadow should be got, and it is generally very desirable to radiograph both elbows for the sake of comparison. Without this precaution imaginary injuries and abnormalities are apt to be discovered.

Of the value of *crepitus* as a symptom nothing needs to be said. But in most cases it is of all symptoms the last that should be sought for. In fractures with retraction it is sometimes not to be obtained. In complete fractures through the shafts of bones or into the middle of joints it is apt to thrust itself on the attention as soon as the limb is handled.

There is a mode of diagnosing the precise position and relations of a fragment which I never see recommended, though it is very obvious,

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and can be made very safe and not more painful than feeling for crepitus. I mean *probing with a sharp-pointed needle.* It should be sterilised and passed through sterilised skin, or through the wound if one exists. Very little anatomical knowledge is required to avoid doing harm with it. I have sometimes got most useful information by this means. It is more accurate and less deceptive than are the X rays.

Now to use the needle as a substitute for inspection and palpation would be rather rough. Its proper use is supplementary, and as a check to the X rays, or substitute for them when they cannot be used.

To give an example: a skiagram suggested that two fragments barely touched each other. The appearance of the limb contradicted this. A few punctures with a needle demonstrated that the fragments were in excellent contact, and greatly relieved both my mind and the patient's.

Any straight needle suffices if long enough. The sharper it is the better. If a handle is required, it can be extemporised by gripping the eye end of the needle with hæmostatic forceps. Or a harelip pin can be used.

In speaking of fractures of the elbow I have not mentioned what should be one of the first steps to take in going into the question of a supposed fracture of the humerus, namely, the step of finding out to what extent the patient can move the limb. When the humerus is completely broken across, the limb is usually helpless, and the patient supports the broken arm with the hand of the sound one.

Chief varieties of fractures in or near the elbow-joint.—To enumerate all would be to unduly burden the memory of any one but a specialist. Of the *humerus*, distinguish fracture (1) of the external condyle, (2) of the internal condyle, (3) T or Y-shaped fractures, (4) fractures of *points* of bone, such as the tip of either condyle, (5) transverse fracture above the joint.

I need scarcely add that the lines of fracture are not generally perfectly straight or rectangular.

Treatment.—Till recently surgeons have generally had each his own favourite plan, which he regarded as applicable to all these varieties of elbow fracture, with slight modifications in one or two cases. The differences of opinion were generally (1) as to the precise angle at which to fix the elbow, (2) as to when passive movements should be commenced. There is something to be said in favour of Robert Jones's plan of fixing the elbow at an acute angle with the hand high up near the opposite sterno-clavicular articulation, even though the ultimate limitation of movement is more likely to be in the direction of extension than in that of flexion. A slight limitation of extension is less inconvenient than a slight limitation of flexion.

With regard to the question of early *v.* late commencement of passive movements, I think that as regards the range of movement to be expected in a given case ultimately, it does not matter whether the movements are begun in a fortnight or in a month. Why should it?

But in a case of slight injury, with little or no tendency to displacement of fragments, postponement of movements of course means delay in attaining full mobility.

Take any given case of fracture into the elbow-joint, and it will most likely be months, even many months, before the full range of movement destiny intends for that case will be attained. The extent of that range depends far more on the accuracy of the coaptation of the fragments than on any plan of movements. I replaced and sutured accurately and firmly an external condyle, which, with the capitellum, had been broken off. The patient was a boy. He refused to permit *any* movements to be made,—in fact, ran away from hospital. I saw him a few months afterwards with perfect mobility in every normal direction.

When a fracture has not been correctly reduced, and movements are painful, I believe they can be so employed as to increase adhesive inflammation and do harm. Patience should be employed. Time tends to round off sharp corners and awkward angles, even when they are inside joints, especially if the patient be a child. In older people there is the danger of rheumatoid arthritis, which appears often to attack injured joints.

I need say no more to demonstrate *the vast importance of accurately reducing fractures into or near joints. The ideal to aim at is replacing the fragments as if they were pieces of a puzzle.*

The prime indication is not simply to put up the fracture in the right kind of splint or in the best position. What is the good of putting a Colles' fracture up with Carr's splint, Gordon's

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splint, or a pistol-shaped splint, according to the rules of which examiners are so fond, if the fragments have not been adjusted beforehand? There are few fractures whose adjustment can be expected to result from the mere application of a splint.

The first means to employ are generally *extension and manipulation*. I need scarcely point out how the simultaneous use of the X rays can help the surgeon to judge of the effect of his manœuvres. But they are not generally available. Sufficient can generally be learnt by inspecting the shape of the joint and testing its movements.

Take, for example, a case in which the external condyle and capitellum are broken off the humerus, pushed upwards and bent forwards, but the ligaments of the joint not seriously torn.

The line E I, instead of being nearly at right angles to the axis of the humerus, will cross it obliquely, the external condyloid ridge will be felt running more forwards than normal, and, when the elbow is flexed, the forearm will move in a plane which slants across the median antero-posterior plane of the upper arm.

All these abnormalities should disappear when the arm is fully extended and supinated.

In such a case as this I should unhesitatingly fix the limb in a position of full extension and supination until union was firm.

If extension and manipulation fail to correctly reduce a fracture into a joint, the question of operation must be considered.

The question of operation.—I am sure you will all agree with me that there are two personal factors to be considered. There is (1) the obvious bearing of the patient's age, occupation, and health, especially the health of his excretory organs, very especially that of his kidneys. But (2) there is also the special experience of the person who will have to operate, should an operation be decided on.

There is nothing mysterious or particularly difficult about the open treatment of fractures into joints. Nevertheless many calamities occur owing to failure to avoid sepsis; and no one should undertake this class of operations in whom the practice of asepsis or antisepsis has not become a second nature, an ingrained habit.

Perhaps each of us thinks himself perfect, but there is one simple test by which we can judge our neighbours with fair accuracy. Do not trust the man (or nurse) who forgets that *time* is an essential element in every asepticising process; no one should think he can prepare his hands for a serious operation in half a minute, then meddle with anything he chooses, and finally repurify his hands by dipping them for a second into some antiseptic solution. Nor should the assistant expect even a careful and thorough preparation of his hands to last all through a long operation, without any attempt to keep up or renew the purification.

Returning to the elbow for an example of a fracture in which a first-class result could not possibly have been obtained without operation, this will be found in a case of mine of fracture of the external condyle, in which the fragment was doubly rotated so that the fractured surface lay beneath the skin while the upper end presented downwards. It was replaced and wired in good position. This case was that of the boy I have already mentioned as refusing to submit to any passive movements whatever. Nevertheless complete range of movement returned in a few months.

Treatment of Tetanus.—Dr. L. Championnière considers the intra-cerebral treatment without danger, and would willingly employ it in serious cases. As for amputation, he has found it useless. The preventive treatment had been efficacious when an epidemic of tetanus broke out in his service. Dr. Girard thinks intra-cerebral injections should not be condemned. There is some danger in using them when the injection does not penetrate into the ventricle, but, on the other hand, this treatment seems to act when the ordinary injections have failed. Dr. Reboul says that he used to see four to five cases of tetanus yearly until 1897. Since then he had made preventive injections of serum, and had only lost two patients, who had not been injected. Dr. Schwartz insists on the importance of making preventive injections. From 1890 to 1898 Dr. Schwartz has seen 315 suspicious wounds in his service, and lockjaw came on in eight cases, with six deaths. Since 1898 Dr. Schwartz has seen 300 cases of wounds received in the street, and there have been only two cases of tetanus—one in a patient who came in with symptoms of lockjaw, and another in one who had not received any injections.—*Medical Record*, Jan. 3rd, 1903.

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WITH DR. E. W. AINLEY WALKER AT GUY'S HOSPITAL.

GENERAL PATHOLOGY OF INFECTION—V.

FOR the destruction of bacteria or other foreign cells which gain an entrance into the body of an animal the interaction of two substances is necessary—namely, the immune body and the complement.

The former of these substances—the immune body—is, as we saw last week, a specific product formed under similar conditions to those which regulate the production of the anti-bodies in general.

In contrast to the immune body the complement, or addiment as it is sometimes called, is not a specific product, being already present in a normal serum. Though not specific, Ehrlich believes it special in the sense of belonging peculiarly to the serum of the animal in which it is found. And it is not indifferently and universally replaceable, though in many cases it is replaceable by the complement obtained from animals of other species than the one used for experiment.

Further, for one and the same serum the complement is not necessarily the same for all the anti-cellular and anti-microbic reactions possible to that serum. Thus Ehrlich has proved in certain cases the existence of two complements in goats, the one extremely labile and destroyed rapidly by a temperature of 56° C., the other still resistant at a temperature of 65° C.

By its action the complement produces the disintegration and solution of the cells which it attacks, and exhibits characters akin to those of the class of bodies which are known as ferments. Professor Ehrlich has put forward the following view of its position and function in the normal animal :—The complement is a ferment produced and discharged into the blood by certain cells—which are not further specified—to serve the purposes of general cell-metabolism. The body-cells possess a group of side chains which exhibit strong affinity for the complement. The complement is therefore taken up out of the plasma by these so-called complementophil side chains, according to the requirement of the moment. It thus enables these cells to carry on their normal metabolic processes by its power of splitting up the large nutritive molecules extracted from the blood, and thereby rendering their assimilation possible.

Now, starting from the position of Pfeiffer that both the substances necessary for protection are present in an immune serum in an inactive form and become active on injection into the body of a living animal, though, as I told you, the complement is present (when present at all) in limited and much less amount than is the immune body, it is evident that it should be possible to supply sufficient of both bodies to ensure protection by giving large enough injections of the serum. But this is not the case, for Roux and Vaillard showed in severe tetanus infections that an animal treated with immune serum in large quantities may rapidly succumb, although it has received such an excess of serum as to render its blood and body fluids capable of protecting other animals against a moderate infection with the same bacterium. And the same facts were proved by Pfeiffer, Wassermann, and others in the case of various other bacterial infections.

And seeing that of the two protective substances the immune body is obviously present in sufficient quantity—since the serum of the dead animal is found to have protective power for others—Ehrlich attributed these phenomena to a deficiency of the complement. Accordingly he was driven to the conclusion that the complement injected in the protective serum is useless to the animal concerned ; in other words, that, with certain not too numerous exceptions, an animal can only make use of its own complement, which is limited in quantity, or, at the most, of complement derived from other animals of its own species.

The exceptions, therefore, became of great importance ; for only by a careful search could there be found for each particular species of animal that foreign complement which could suitably replace the deficiency of its own proper complement and bring about the destruction of the invading micro-organisms. And more especially for serum-therapy in man Ehrlich regards this search as of prime value for the immediate future.

I am myself unable to agree with Professor Ehrlich's view of the extreme specialism of the complement to its own species of animal. That his position is by no means satisfactory is shown by the following facts. In every observation of Pfeiffer's phenomenon made by means of the

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immune sera of other animals in the peritoneal cavity of the guinea-pig, the school of Pfeiffer holds that the inactive complement of this serum is rendered active by the agency of the endothelial cells. That is to say that a foreign complement can be made use of by the guinea-pig so that it can now, even after its removal from the peritoneal cavity, exhibit its activity against the bacterium concerned. Again, Wassermann has shown that in infections of the guinea-pig a satisfactory complement, both for the animal and for the immune body of dog's serum, which he used for the specific protection, is present in fresh serum of the ox—an animal belonging to a species widely different both from the guinea-pig and from the dog. Ehrlich himself has also found a number of exceptions to the law which he proposed. And I have shown that in the case of guinea-pigs treated with horses' immune serum for typhoid infection, suitable complement can be supplied from the fresh serum of the rabbit, ox, and pig.

Complement, therefore, does not seem so special to the species as Professor Ehrlich thought. The deficiency of complement observed in animals infected with large doses of bacteria, and treated with specific immune sera, is the result, not of an inability of the animal to employ the foreign complement, but of the fact that the specific immune serum injected contains no complement of any kind at all, but only the specific immune body.

The complement is not simply inactive in the immune serum, as was taught by Pfeiffer, but it is absent. It is so extremely labile that it disappears with great rapidity when the blood is shed. And I have watched this disappearance of the complement from the fresh blood of animals from the actual moment when the blood is shed, until its presence is no longer recognisable. After its disappearance it can be replaced from the fresh serum of various other species, whether the experiments be made in test tubes or in the body of a living animal.

I have already told you what is the view of Ehrlich as to the nature and origin of the complement, which he considers to be a normal metabolic ferment formed by certain cells by which it is discharged into the plasma, there to circulate until such time as it is taken up by other cells, which it assists in carrying on assimilative processes. This view, however, has now become untenable on various grounds. These we shall next examine.

Just as we saw to be the case in the destruction of bacteria, so in hæmolysis of red corpuscles which have been introduced within the body of an animal, Metchnikoff has shown that phagocytic action plays a definite and increasing part with increase of the animal's immunity, until when full immunity has been acquired the phagocytic method of destruction may be almost universal and exclusive.

The school of Ehrlich also now admits that the destruction of bacteria or other cells can take place intra-cellularly within the phagocytes as well as extra-cellularly in the body fluids. Now such intra-cellular destruction must either (1) pursue a course entirely different from the extra-cellular, or (2) it must be occasioned by the same substances as cause the extra-cellular lysogenesis. And in this case the necessary immune body and complement would on Ehrlich's theory have to be taken up by the phagocytes from the blood-plasma to enable them to carry out the process of destruction.

1. The former of these two alternatives would lead to the remarkable position that the animal body, while possessing a basis of protection in the natural biological process seen in the universal tendency to phagocytic action, yet has resort in the acquirement of immunity to the development of a purely chemical reaction in addition—that of the immune body and the complement; and, not content with this, develops and increases equally the more complex biological method of ingestion and destruction by the phagocytes, with the result that at a period when the chemical reaction has reached a maximum it is actually found to be entirely superseded by the intra-cellular method of destruction. That is to say that the bacteria or other cells injected are taken up from body fluids admittedly capable of causing their solution and destruction to be destroyed by other methods by the phagocytes. This is absurd, and renders the immunity reaction meaningless.

We are, therefore, forced to the conclusion that the two processes of intra- and extra-cellular destruction are not different from each other and independent, but that they are identical in nature.

2. The question then arises, Do the phagocytes really obtain the complement and immune body which they require to bring about the lysogenesis from the blood-plasma in which they circulate? If this were so, phagocytosis would present itself

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merely as an epiphenomenon in the process of protection of no especial value and of no particular importance. And it would be impossible to explain why, as immunity increases, living and active bacteria, for example, can be shown to be more and more eagerly ingested by the phagocytes and then destroyed, when this result could be obtained as easily and more conveniently by their remaining in the plasma, which contains (on Ehrlich's view) both immune body and the complement.

But as a matter of fact the serum of animals which are naturally immune has been shown to contain no recognisable amount of immune body, and yet in these the phagocytic destruction of bacteria is exhibited in its most active form. It follows that the phagocytes must themselves contain the receptors which in immunised animals are discharged into the plasma as the immune body, and which are necessary to initiate lysogenesis.

It, might, however, still be possible that the complement is a free ferment, which the phagocytes obtain from plasma. Let us examine this possibility.

We have the following facts:—Anti-microbic serum possesses *in vitro* no bacteriolytic action, but it does contain the immune body; the other necessary substance—the complement—is therefore absent. Such serum is bacteriolytic when freshly obtained from the living animal; the complement, however, is very labile and soon disappears. It may be restored by the addition of fresh normal serum (Bordet), or of leucocytic fluids (Hahn); and Bordet showed that a definite relationship exists between the mass of leucocytes added and the degree of bacteriolytic power obtained. That is to say, the amount of complement is in relation to the number of leucocytes added. Again, Denys and Havet discovered that a bacteriolytic pleural exudate loses its activity entirely on the removal of its leucocytes, but that its power can be immediately restored by merely putting back the leucocytes.

Moreover, I have found the following facts:—(1) That a deficiency of complement in guinea-pigs can be made good not only by the injection of fresh normal serum, but also by an extract of fresh blood-clot from which the serum has been separated. Complement, therefore, is present in the clot as well as in the serum. (2) That serum when it first begins to separate from the clot contains only a little complement, but that this rapidly increases in amount during the first few hours after the blood is shed if the serum which is separating be left in contact with the clot, while if the serum be removed into a separate vessel no increase in the amount of complement occurs. And Bordet and Gengou have recently proved that plasma which has been obtained by special methods that prevent the breaking down of leucocytes in the shed blood contains no complement and has no lytic action on bacteria at all.

From all these observations it follows that the complement is not a ferment circulating freely in the plasma, as was held by Ehrlich, but is exclusively a phagocytic product, only set free into the circulating plasma or the serum by the disintegration of the phagocytes themselves.

The intimate relation of the complement to leucocytes has been established in a different manner in some experiments by Dr. Beaton and myself. Rabbits were immunised to a particular bacterium; their blood then contained the immune body for this micro-organism. They were then treated with another bacterium in order to produce leucocytosis. This did not affect the immune body for the first bacterium, which remained approximately constant in amount; but it was found that the bacteriolytic power of the blood varied from day to day exactly with the number of leucocytes present, and reached its maximum exactly at the height of the leucocytosis caused by the injection of the other bacterium. The amount of complement present accordingly depends entirely on the number of the leucocytes. Complement, therefore, is, as Metchnikoff maintains, the ferment of the phagocytic cells.

Where the animal is immune or has been immunised the complement performs its function of bacteriolysis within the body of the phagocyte, which is its normal situation after the bacteria have undergone ingestion; but where the animal is more susceptible and phagocytes are destroyed by the bacteria it is set free into the plasma, and can there also carry out bacteriolysis. It only acts, however, as I have told you, after the immune body has attached itself to the bacteria.

This immune body, as we saw, is also present in the phagocytes. Whether the animal is naturally immune or not depends, in my opinion, only on

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the amount in which it normally exists within these cells. The effect of an immunisation is to stimulate these receptor groups, which multiply and become split off into the plasma as free immune body.

The formation of the immune body by the leucocytes is proved by the following observations. The experiments of Deutsch, confirmed in general by the results of Metchnikoff, Wassermann, and others, led him to the conclusion that in bacterial infections the immune body is formed in the tissues of the spleno-medullary system, which are, of course, the seat of the formation of the blood-phagocytes. And whenever it is being formed in quantity the bone-marrow exhibits evidence of markedly-increased activity, as shown by Muir and others.

Moreover, Bulloch found for hæmolysin that the curve of immune body present in the blood follows the curve of daily variation of a particular variety of leucocytes.

Whether it is formed exclusively by leucocytes, as Metchnikoff believes, or is produced by other cells as well—the view maintained especially by von Dungern—is not perhaps at present quite determined. There is, however, no evidence obtainable of any marked activity in other tissues when it is being formed. Nor is there any reason why an anti-body formed by other cells should hold the very high affinity for complement; that is to say, for the ferment of the phagocytes which is exhibited by the immune body, since evidence is altogether wanting of any close relationship between the intrinsic ferment of the phagocytes and the side chains of body-cells in general.

We see, then, on the whole that the destruction of bacteria or other foreign cells within a living animal is brought about by the interaction of two substances produced by phagocytes—namely, a preparatory substance or "preparer"—immune body—and a digestive ferment which destroys the foreign particles, but which can only act when the "preparer" has already played its part. In animals which are immune or immunised this process is accomplished in the bodies of the phagocytes, which rapidly ingest the living particles; but where the animal is more susceptible destruction of the phagocytes themselves sets free these substances into the plasma and leads to extracellular attack on the infective agent.

There still remains one phenomenon associated with the reaction of immunity which is of great importance—I mean the phenomenon of agglutination.

Charrin and Roger found in 1889, in the course of their experiments with the bacillus pyocyaneus, that this bacterium when grown in its specific immune serum showed the remarkable peculiarity that it collected into clumps and was deposited at the bottom of the culture tube. This observation was confirmed by Metchnikoff for certain other micro-organisms. He showed, however, that it does not occur with all bacteria. Accordingly it could not form a necessary part of the immunity reaction in all cases; and Issaeff found that it does not entail any impairment of the vitality or virulence of the bacteria which are thus agglomerated.

Bordet was the first to show that not only do bacteria which are grown in their specific serum thus agglomerate, but also fresh bacteria introduced into serum give the same reaction. This phenomenon is now usually spoken of as agglutination. And Pfeiffer found a similar process to occur in the destruction of cholera vibriones within the peritoneal cavities of guinea-pigs. Then came the discovery made by Herbert Durham in Gruber's laboratory in Vienna, and worked upon by Gruber, Durham himself, and Grünbaum, that in recovery from typhoid infection the blood acquires agglutinating action on the bacillus typhosus. It happened that the application of Durham's reaction to the serum diagnosis of typhoid in man was published first in Paris by Professor Widal. The credit of the discovery is, however, due to Durham, and here in Guy's, at any rate, the reaction ought to bear his name.

As the result of their investigation of agglutination Gruber and Durham came to the conclusion in 1896 that in agglutinative immune sera there are present neither substances which destroy bacteria, nor such as stimulate the body-cells to carry out bacteriolytic action, but that the action of the agglutinins, or substances which cause agglutination, is to induce a swelling up and alteration of the bacterial envelope, which is thus rendered permeable by the bacteriolytic substances. Upon this view agglutination is a necessary antecedent of bacteriolysis with the agglutinable micro-organisms—such, for example, as the bacilli of

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typhoid fever and of glanders, the bacillus pyocyaneus, the bacterium coli, and the cholera vibriones.

On the other hand Malvos endeavoured to throw doubt upon the origin of the agglutinins, and questioned whether they arose from the reaction of the animal at all. Thus he found that an agglomeration of bacteria could be induced by the action of certain chemical substances, such as formalin, corrosive sublimate, saffranin, vesuvin, fuchsin, acetic and lactic acids, and other reagents. Subsequently he showed also that culture media in which the bacteria have grown contain similar agglutinating substances. He therefore held that the agglutinins of blood have simply been derived from the injected cultures, and are not anti-bodies formed within the animal during the process of immunisation.

This view of Malvos is untenable on the following grounds: The agglutinins are discharged freely in the secretions of the animal concerned (Nicolle), and yet the agglutinating action of the blood may be retained for years after the animal has been immunised. Moreover, just as in the case of other anti-bodies, an animal may be frequently and very freely bled without a permanent reduction of the agglutinating action of its serum; from these results it follows that agglutinin must be produced within and by the animal itself. This I was able to show conclusively in the following experiment:

Of two rabbits among a number employed in certain observations, the one had received on seven successive days a certain intra-peritoneal injection of living typhoid culture, while the other had received the same infecting doses, and at the same time on each occasion 5 c.c. of highly agglutinative anti-typhoid serum. Now it is evident that if the view of Malvos were correct and the agglutinins were derived exclusively from the bacterial cultures introduced, the serum of the animal which had received both culture and agglutinating serum should have become more highly agglutinative than the serum of the other animal, which had been treated with the cultures only. As a matter of fact, the serum which it yielded was only about one half as agglutinative in its action as the other. It follows, therefore, that the agglutinins are formed by a reaction of the animal, and consequently that the animal which was in part protected by the immune serum given reacted less and formed much less agglutinin than did the unprotected animal.

Agglutinins are, therefore, anti-bodies, and Deutsch concludes from his experiments that they are formed within the tissues of the lung. Deutsch's results have been confirmed by an Italian worker named Moreschi. Ruffer, however, has lately found a close relation between the daily leucocytic count and the acquirement of agglutinative action during the process of immunisation, and he concludes that the agglutinins are formed by leucocytes.

The view of Gruber and Durham that agglutination is an essential antecedent of bacteriolysis for the particular bacteria concerned was strongly opposed by Pfeiffer, Metchnikoff, and others: and Pfeiffer and Kolle showed that in the cholera peritonitis of guinea-pigs protection can be carried out without the occurrence of agglutination. Similarly Bordet found that both agglutinative action and protection could occur each in the total absence of the other. And these results have been confirmed by various workers.

But Durham has produced considerable evidence of the existence of a very close relation between the phenomena of agglutination and protection. Thus he showed that an increase in the protective action of a serum is accompanied by corresponding increase in its agglutinating power, and that complete agglutinating power, complete protection, and complete bacteriolytic action are associated. Deutsch further found that the agglutinins and protective substances develop simultaneously, both appearing, both reaching a maximum, and both again gradually diminishing in quantity in the same number of days from the injection of bacterial cultures.

Again, I found in the examination of a number of anti-typhoid sera against a number of varieties of the bacillus typhosus, that the agglutinins are, like other anti-bodies, not only specific for the bacterium in question, but peculiarly "special," as Durham termed it, for the particular variety of that bacterium which gave rise to their formation; that the protective action of a given serum for any given variety of the bacillus typhosus was in proportion to its agglutinating power for that variety; and that the agglutinability of any given variety varied inversely as its virulence.

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Hence the two processes of agglutination and protection follow clearly parallel though not superposable directions. I say not superposable, because in the gradual loss of an acquired immunity some degree of protective action may remain at a time when the agglutinating power has disappeared.

The agglutinable substance of bacteria which agglutinate resides apparently in the bacterial envelope, and is diffusible. It certainly diffuses out of the bacteria into the culture media in which they are grown, for media out of which the bacteria have been filtered will, on the addition of agglutinating serum, exhibit changes comparable to agglutination. That it resides in the bacterial envelope was proved by Kraus. And Harrison found that it can be digested and removed by the action of the pyocyaneus ferment leaving the bacteria non-agglutinable. That the reaction of agglutination is independent of the life of the bacteria follows from the observation that dead bacteria both cause a production of agglutinins after their injection in the living animal and are themselves equally agglutinable with their living representatives.

The nature of the relation which exists between the agglutinative process and protection is quite unknown. But I suggest to you that it may possibly assist phagocytosis, and that just as the chemical reaction of bacteriolysis is aided and initiated by the action of the immune body, so the preliminary physical ingestion by the phagocyte may perhaps be assisted for the bacteria concerned by the occurrence of agglutination.

This is hypothesis; but if it were established it would supply an explanation of the fact that the agglutinative and protective action of an immune serum are not coincident though they run parallel. For the protective action is made up of two constituents, namely, the bactericidal and the antitoxic action of the serum, while phagocytosis only deals with one of these, the former. And hence the agglutinative process would have no particular relation to the antitoxic, but might be quite coincident in its development with bacteriolytic action.

It follows, then, from all that we have learned of the processes occurring in infection, that the reaction of the animal body to bacterial invasion is developed along two lines, the first directed to the destruction of the invading micro-organisms, the second to a neutralisation of their products. The latter, which we broadly term the antitoxic action, concerns all dead bacterial substances whatever which can be taken up by body-cells, and leads to the formation of the anti-bodies, which include the antitoxins proper, agglutinins, coagulins, the immune bodies and the like. These anti-bodies are liberated side chains or receptors of those cells which are respectively susceptible to the bacterial products, and the immune body consists of such side chains derived from phagocytes.

The antitoxins neutralise the bacterial toxins, and thus prevent intoxication of the animal concerned. And the bacteria are themselves digested by the phagocytic ferments. The action of these ferments is made possible by a preliminary union of the immune body with the bacteria concerned.

On the destruction of the phagocytes their ferments are set free into the plasma, and can there perform their function of bacteriolysis; but in immune or immunised animals bacteriolysis occurs within the bodies of the phagocytes after the ingestion of the micro-organisms.

Similar facts are true for micro-organisms other than bacteria, and for other foreign cells.

Successful resistance to infection, therefore, means the possession or formation of specific anti-bodies in sufficient quantity, and the supply of the essential lysogenetic ferment by a proliferation of the cells which form the phagocytes. Failure of either of these processes entails a fatal termination of the infection.

Next time I shall show you what specific treatment is available for man, and then commence the pathology of fever.

Osteo-arthritis Deformans following a Sprained Knee.—Deshusses reports the case-history of a man æt. 32, who fell, injuring his left knee. Swelling followed, with hæmarthrosis, which was treated by puncture, aspiration, and compression. Pain persisted in the knee, and in eighteen months the deformity of the knee was marked. The knee-joint was resected for the osteo-arthritis deformans, with excellent operative result. The diagnosis was not difficult. Nothing in the treatment was effective until resection was performed. (*Journal des Sciences Médicales de Lille*, October 4th, 1902).—*The Philadelphia Med. Journ.*, December 27th, 1902.

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A LECTURE
ON
SOME POINTS IN THE PROGNOSIS OF HEART DISEASE.

Delivered at the Medical Graduates' College and Polyclinic.

By P. H. PYE-SMITH, M.D., F.R.S.,
Consulting Physician to Guy's Hospital.

THERE are few subjects which come more home to our bosoms and business in daily practice than affections of the heart, and nothing more important from the patient's view than their forecast.

I suppose the first question when we have a case of valvular disease with a murmur to be heard, is whether this murmur is functional or organic, and those who are most experienced will, I am sure, agree with me that in some cases the question is extremely hard to decide; in fact, compared with diseases of the lungs, most cardiac diseases are difficult. There are seldom two competent and experienced men who, in a case of bronchitis, empyema, or phthisis, would seriously differ from one another; whereas there are few cases of cardiac disease in which two men equally experienced and capable might not differ. One reason for this is that we are too apt to assume our task to be easy, and all the more because some cardiac lesions are no doubt readily recognised—for instance, the to-and-fro basal bruit and the water-hammer pulse of aortic regurgitation, or a presystolic murmur with a thrill at the apex; and in both cases the physical sign is certain in its meaning. But we sometimes err by not giving ourselves sufficient time and trouble. I have seen a man listening to a cardiac case when he has been himself in so inconvenient a position, leaning over the patient's bed and supporting himself with an effort, that one is quite sure his mind must be half taken up with maintaining his own centre of gravity. If, instead of leaning over the bed, he would kneel down and keep his mind entirely on

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the auscultation, the character of the sound would often become plain enough. Or, while listening to the heart, one's attention may be interrupted by some trivial remark of the patient's—we all know how some will still be talking—and one cannot listen to the patient and his heart at the same time. Or one's eye is caught by the patient's lividity or pallor, and one's attention is no longer auditory but visual; we hear, but we do not listen. The only remedy is patiently to go back and listen again. What was obscure will then become clear, especially after feeling the pulse. The diagnosis, then, between functional and organic diseases often requires deliberation and attention, and repeated examinations. It is no disparagement to a man that he reserves his judgment until a second time of asking.

Certain rules will help us in the interpretation of the sounds heard. One is that we never—but in medicine we must not use the word "never" one day, lest we be contradicted on the next—we "hardly ever" meet with a diastolic murmur which is not organic. And the same rule applies to the presystolic murmur. Again, an apical systolic murmur heard well round in the axilla, especially at the angle of the left scapula, is an almost absolute sign of an organic lesion. In most cases we are much helped by the pulse, and that often will decide the question whether a systolic murmur is functional or organic. Moreover there are certain probabilities which justly weigh with us. We know it is very rare for an organic disease of the valves to be idiopathic or primary without an ascertained origin or cause; and it is when we know thoroughly the pathology of cardiac valvular lesions that we gain our greatest help in distinguishing between functional and organic murmurs. A history of true rheumatism, including chorea and post-scarlatinal synovitis, the large inelastic pulse of atheroma, the signs of early senile changes, the presence of traces of syphilis, the history of recent enteric fever, of pneumonia, or of suppuration in the systemic circulation, become important helps to diagnosis by the well-tried road of pathological probability.

While the patient is standing up we are sometimes doubtful whether there is a murmur or not; but if we make our patient lie down, and on listening again at the apex we hear a murmur quite unmistakably, the fact is, I think, a good proof that the bruit is organic and not functional. A functional murmur is to be heard when the patient is on his feet, and still more loudly when he has been hurrying upstairs or coming into our room with trepidation. An organic murmur is best heard when the patient is quiet and lying down.

When we are satisfied that a murmur is organic, *i.e.* is caused by a fluid vein due to a permanent structural change in the valve, what is to guide our prognosis? Many years ago the following order of gravity was put forth by the late Dr. Peacock, whose portrait I am glad to see here, for he was a man who ought to be remembered, not only for the accuracy of his observations, but for his scrupulous consideration for the humblest and the youngest of his colleagues. The least serious is aortic obstruction, next comes mitral obstruction, then mitral regurgitation, and, most dangerous of all, aortic insufficiency with regurgitation. I believe this to be true as far as it goes. But then it is modified by the co-existence of more than one lesion, and by the state of other organs, particularly the arteries, the kidneys, and the lungs. The prognosis of valvular disease when more than one orifice is affected, follows the graver lesion of the two, *i.e.* the aortic rather than the mitral incompetence, and the obstruction rather than the concomitant leaking of the mitral valve. Most cases of a contracted mitral valve end with more or less regurgitation; all aortic cases, if not cut short, end first in mitral and then in tricuspid regurgitation. Contraction of the tricuspid valve is very seldom found without contraction of the mitral also, and does not, when recognised (which is not always a matter of course), appreciably affect the prognosis.

Of distant organs, the most important are the kidneys and the lungs. If the kidneys are healthy we can use mercury and digitalis, and, if needful, opium without scruple; if they are diseased we are debarred from or hampered in using these powerful drugs. But when with albuminuria the specific gravity of the secretion is high, the amount small, and the colour deep, with deposit of urates and perhaps a trace of bile, we may assume that the kidneys are congested, not permanently diseased, and we may rely upon their help.

Bronchitis, whether acute or chronic, is of unfavourable effect on cardiac cases; pneumonia less so, and phthisis scarcely at all.

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A most important element in prognosis is the origin of the cardiac lesion. Whatever orifice is affected, if the process began with rheumatism (including scarlatina and chorea) the prognosis is better than when the origin is atheromatous, and begins in the ascending aorta.

Patients with rheumatic endocarditis are almost always young, and heart disease is one in which on the whole we may say that the younger the patient the better the prognosis. It is a pregnant and remarkable fact that true rheumatism is a disease of youth. The older a man gets the less likely he is to become rheumatic, and if he should be attacked he is less likely to have his heart affected. The prognosis of diabetes is the reverse; in children, happily, very rare, but rapidly fatal; in young men diabetes is still a terrible disease. As our patients advance to middle age we can give them good hope under proper treatment; and at seventy years we should often overlook diabetes but for the routine practice of examining the urine for sugar.

If rheumatic endocarditis is the most favourable origin of valvular disease, and atheroma much less so, a congenital origin takes the third place, and septic endocarditis is the most dangerous of all. Among the most dangerous cardiac cases are those with all the symptoms of mitral incompetence, the dyspnœa, the dropsy, the emaciation, the large liver, the weak and irregular pulse, but no mitral murmur. These are mostly met with in children; they follow rheumatism and depend on an adherent pericardium. They are very little amenable to treatment.

Of the general prognosis of cardiac disease, I would say first that we form our forecast not so much by the physical signs which lead us to the diagnosis, but by the absence of symptoms. We see, every now and then, cases which, until by accident we investigate the heart, give us no warning of the serious lesion that is present. The patient does not complain of his heart, and none of his symptoms point to it; in fact, we may almost say that a patient who does complain of his heart, who is sure his heart his wrong because there has been pain and palpitation, is probably suffering only from flatulent dyspepsia, or from some form of neurosis. On the other hand, the pain that does not speak is that which is most severe and dangerous. The patient with real cardiac disease comes to you complaining of asthma, or indigestion, or headache, or cough, or a bilious attack. First, then, we inquire into the symptoms. If a patient can walk some distance; if, especially, he can go uphill and upstairs; if he can exert himself moderately without distress; and if when you set him to walk up and down the pulse is not much accelerated and continues regular; then, however loud his murmur may be, the prognosis is comparatively good. He has, as we say, good compensation. The proverb is: "If the axe is blunt you must put to it more strength;" and if the pump is leaking it requires more strength to get the amount of water necessary. That is what we mean by compensation of the heart; when the valvular mechanism is imperfect the muscular force-pump of the left ventricle must do more work. In the case I have been supposing, the increase of strength is small, and it is sufficient; the patient is free from the symptoms of severe cardiac disease. Some will say it is a good thing, in a case of aortic (sigmoid) insufficiency, to find that the left ventricle is very much enlarged, that the impulse is heaving and strong, the apex displaced to the left, and the dulness increased. No doubt it is better that the patient should have hypertrophy than that he should not have the valvular fault compensated. But we must not suppose that that is the best thing; the best possible is for the hypertrophy to be so very small that it does not show itself by any physical examination. For the patient who does not show symptoms of cardiac disease, in whom there is no dyspnœa or dropsy or jaundice, prognosis is surely the better if we do not find any palpable evidence of compensating hypertrophy. The worst prognosis is when we find that compensation has taken place; that a patient for a long time has been pursuing quiet occupations in considerable comfort, as many do, or filling laborious occupations even, as a few do, that at last compensation has broken down; that what was hypertrophy is now dilatation; that the muscular walls, which were thick and strong, are now flabby, and perhaps beginning to undergo fibrous degeneration; in fact, that the very process which was compensatory during the good health and comparative youth of the patient, now that the tissues are inferior to what they were, and the patient is getting old, leads to degenerative changes. Such degeneration will probably affect the hypertrophied skeletal muscles even in a healthy and athletic man, if training is kept up too severely or

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too long. Or it may be that the valvular lesion has become more and more pronounced, so that the increased force which has kept things going is beginning to be unequal to the need, and the fatal circle has been entered on which will, unless broken, end in cessation of the circulation, and the equilibrium of death. This process of hypertrophy of the left ventricle is therefore a consecutive process; but the best prognosis is when only a slight degree of it is sufficient. It may be accompanied—probably with advantage—by some degree of dilatation, but if replaced by that change the prognosis is not better but worse.

Hypertrophy is a physiological process, as we see in a loaded frog's muscle in the moist-chamber, as we see in the blacksmith's arm, in the œsophagus above a stricture, in the colon above a tumour, and in the bladder above a contracted urethra. Dilatation is a physical result of pressure, as we see in an old shoe or an old glove. In cases of valvular obstruction, hypertrophy is the active response of a well-nourished, elastic, muscular cavity, dilatation the effect of mechanical internal pressure on the walls of a yielding cavity.

In forming a prognosis, then, in cases of aortic obstruction we look to signs of hypertrophy of the left ventricle; in cases of mitral obstruction to the left auricle, where hypertrophy is gauged not by palpation and percussion, but by the loudness, pitch, and duration of the presystolic murmur.

The prognosis of cases of regurgitation through the aortic valve is more grave *cæteris paribus* than in obstruction at that orifice; but in the former case, while hypertrophy is a compensatory process a limited dilatation is probably advantageous.

In cases of mitral incompetence, the left ventricle ought not to be either dilated or hypertrophied, and probably is not unless there is some other cause in the aorta, in the pulmonary capillaries or in those of the systemic part of the circulation.* The natural compensation for mitral incompetence is hypertrophy of the right ventricle; hence a broadening of the percussion-dulness of the heart towards the right, with epigastric pulsation, is a favourable sign, for a strong right ventricle often delays the advent of dropsy and other effects of congestion of the systemic veins for months or years.

A special point of prognosis is the degree of liability to sudden death. Popular opinion associates sudden death with heart disease; and on the whole it is right. It is very rare for an attack of apoplexy, or epilepsy, or angina pectoris to prove instantaneously fatal as cardiac syncope does, and we find that the great majority of cases of sudden death which come to the post-mortem room are cases of organic disease of the heart. What difference is there among the forms of cardiac disease as regards their liability to this sudden and terrible termination? We should all say at once that aortic regurgitation is the one to be dreaded most, and statistics confirm the general impression. Fatal syncope may occur—and perhaps most often does occur—in cases of good compensation, with a well-hypertrophied left ventricle, with little or no dilatation, in persons who go about their work well and are living in considerable comfort. I am afraid those are the most liable to have this favourable course of the disease suddenly stopped by syncope, during which the brain is no longer nourished by blood sufficient to keep it going, and so very rapid and sudden dissolution takes place. If a man has the same lesion of the aortic valves and compensation begins to fail, and his left ventricle begins to dilate instead of being hypertro phied, then there is a relaxation of tension, and the case goes on to the common condition in which all cardiac disease ends—which is not cut short by intercurrent disease or death—just as all affections of the cord tend to one of paraplegia with septic nephritis, and all cerebral diseases to coma, for in the last stages of a journey to one bourne all the roads converge.

This condition, marked by dropsy, a large liver, jaundice, dyspnœa, ascites, and very often effusion into one or other side of the chest, though characteristic of chronic mitral regurgitation, is met with also in the last stages of other forms of heart disease as a secondary effect. Aortic valvular disease in its last stages, when compensation has broken down, assumes the pulse, the aspect, and the symptoms of mitral disease, with comparative immunity from sudden death. The friends and the patient, too, have, I think, the right to know everything we can tell them if it be for their good; and we may tell a man with aortic

* The displacement of the cardiac apex to the left, which is often taken as evidence of hypertrophy or dilatation of the left ventricle in mitral cases, probably depends on enlargement of the right ventricle and left auricle displacing the apex downwards and to the left.

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regurgitation and good compensation that, if he is content to go about quietly and prudently, to keep our rules, and make rules for himself—because no rules we lay down meet every event, and the wise man is he who understands the principle rather than the details of our advice—he may live long and usefully; but that any sudden and severe strain may prove fatal—that he lives on probation. On the other hand, when the compensation breaks down, and when the dropsy and albuminuria and icterus begin to appear, it may comfort the sufferer, and his wife and friends, to be told that the danger of sudden death is much less, partly because he is no longer able to get about and run the risks which he could before. But that is not all, for some of our patients with aortic regurgitation die in their sleep. Another reason is that the process has become different—that there is not so much arterial anæmia, and there is more venous congestion. The first is much less distressing and much more dangerous; the other is most distressing, but it is not so immediately dangerous.

Next to the cases of aortic incompetence come those of mitral stenosis in liability to sudden death. It is very rare in pure mitral incompetence.*

A complication to which patients attach great importance is *hæmoptysis*. I think there is no doubt that the form of heart disease which most often produces this symptom is mitral obstruction. The narrowing of the mitral orifice causes dilatation of the left auricle, and the pulmonary veins having no valves, extreme congestion and often thrombosis occurs in the pulmonary capillaries. Hæmoptysis in aortic regurgitation is also not uncommon. In cases of aortic stenosis and of mitral regurgitation, hæmoptysis is rare, as it is in those of adherent pericardium and of degeneration of the muscular fibres of the heart. This symptom is, however, more important in the eyes of the patient and his friends than in ours. This hæmoptysis, I have no doubt, is often a useful mode of venesection, and the patient is often much better after he has spat up a quantity of blood. I never saw a patient die from hæmoptysis in the course of cardiac disease, and I have never seen one the worse for it. There is no fear of it bringing about phthisis, an ancient view expounded in the seventeenth century by Morton, which was revived by Neumeyer, and afterwards held the stage for fifteen or twenty years. It has now been deservedly sent back to the lumber room of discarded theories. We know that no case of phthisis begins in hæmorrhage, that the hæmorrhage is a mere complication, and that the hæmorrhage of heart-disease is incapable of originating phthisis. On the other hand, my late friend, Dr. Moxon, thought (and he made some beautiful drawings of post-mortem specimens to illustrate the point) that the effect of marked heart disease is rather to retard the influence of tuberculosis when it affects the lungs. This is, of course, difficult to prove, but anatomically we find frequently cases of chronic pulmonary tubercle not making active progress, with fibrous cicatrices and contraction, and with but slight symptoms during life, in cases which prove fatal by coincident heart disease, where there is the narrowing of the mitral valve which most frequently leads to hæmoptysis.

With regard to compensation, I will only repeat that the more it is hypertrophy, and the less it is dilatation, the better, on the whole, it is for the patient. A small degree of dilatation may possibly, in aortic incompetence, be useful, and possibly in the case of mitral stenosis in the left auricle. But certainly, if it is, it is only a very small amount. What we depend upon is the thickening of the walls, the true hypertrophy.

What is the prognosis in cases not of valvular disease but of other organic diseases of the muscular wall, or of disturbed innervation which we are not able to trace to any organic cause?

My own belief is that cases of fatty over-growth, obesity of the heart, where there is a mass of subpericardial fat encroaching on the muscle, and the cardiac wall thinned away in consequence, so that the pericardial layer almost meets the endocardial, are extremely dangerous. They occur in obese and intemperate people—publicans and others—and they often lead to sudden death. On the other hand, fatty degeneration in which there is molecular fatty change in the muscular fibres of the left ventricle causing a "tabby" streaking, is far less dangerous than is generally supposed; and I, for one, am sceptical as to any means of recognising the condition during life. I believe that most of the symptoms that have been supposed to belong to it have been thought out in the study, and have not been brought to the test of the dead-house. The only way in which I can diagnose this fatty

* See Dr. L. E. Shaw's observations quoted by the writer in the 'Text-book of Medicine,' vol. ii, p. 269.

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degeneration of the muscles is not by any physical signs, but by a knowledge of pathology. If you have a patient with Addison's idiopathic (since called pernicious) anæmia, you can be sure that there will be found fatty degeneration of the heart. If you have a case of phosphorous poisoning you can be equally certain in diagnosing the same condition. It is one of those cases in which our diagnosis does not depend upon seeing and feeling, but upon pathological experience. In the case of this molecular fatty degeneration which we see in many cases of valvular disease, I doubt if it would affect the prognosis, even if we knew it was there.

Then there are those cases of disease of muscular fibres of the heart which are characterised by overgrowth of fibrous tissue, which are often syphilitic in origin, so that we find gummata embedded in the walls of the heart. We find at other times syphilitic endarteritis of the coronary branches, sometimes causing thrombosis in the vessels of the heart. These cases, which are somewhat rare, and extremely difficult to be sure of during life, have a considerable pathological significance, and as far as one is able to see from the limited opportunities which most of us have had in watching these cases, they are irremediable by any means at our command, and proceed steadily to a fatal termination. Death appears usually to occur from syncope.

Now let us consider the prognosis when the chief symptom is derangement of the pulse, without evidence of organic parietal or valvular disease. Tachycardia, or a persistently rapid pulse, is a very serious affection. We are familiar with the rapid pulse of emotion and excitement, and the rapid pulse which accompanies mitral, and as a rule most other forms of valvular disease of the heart; but tachycardia, without any apparent reason, persisting for weeks or months or even years, is happily not very common, because, as far as I know, it is a very fatal disease. It generally ends in hypertrophy, sometimes in dilatation of the heart, and not infrequently in sudden death. Such cases have been recorded again and again, and it is remarkable that their origin is quite unknown. They form a group about which one can say very little, either with regard to the pathology, the cause, or the treatment. Then there is the opposite condition—namely, a very infrequent pulse. That is much less common. It is again remarkable to notice how much easier it is for the heart to be accelerated than it is for it to be slowed. Almost every febrile disease, almost every nervous disease, and a great many others, are accompanied by a frequent pulse. We know how easy it is to accelerate the pulse in health by running or by emotion. What a contrast to slowing the pulse—bradycardia! By lying down and keeping cold you may bring down the pulse a little in frequency. You may watch a person who is suffering from starvation, due for instance to stricture of the œsophagus, and you will find that the pulse is somewhat slow. But it is only a few beats below 70, and it is liable to quicken very readily on the slightest provocation. If bradycardia goes down to 60, that is what many people have as a constant condition, with no other sign of heart disease. But when it sinks to 35, or 30, or 28, then this extreme bradycardia, though happily a very rare, is a very dangerous disease. I have only seen well marked two or three cases, and they have gone on from bad to worse. In some of these cases it is remarkable that instead of ordinary attacks of syncope there are convulsive attacks. This has been recorded by Dr. Alexander Morison, in a monograph on the subject; and in two cases which I had under my care I can confirm his observations.

I know of one case in which bradycardia has existed for many years, not without symptoms of dyspnœa, but with prolongation of life to the natural span.

With respect to the bearing of prognosis on the treatment, let us take an average case of well-compensated aortic incompetence. We should not be eager to treat it at all, for the anatomical lesion we cannot touch, and the help our best treatment could afford is already given by the operation of natural process. I do not like to speak of the *vis medicatrix naturæ*, because it is one of the "idols of the theatre" which have passed away. We do not now believe that "Nature" is an intelligent cause of any improvement. As to the intentions of Nature, she has no intentions. Sometimes natural processes will make a disease worse, sometimes better. We cannot count upon an Archæus in the body to protect us. And if we rise to a higher conception of "the course of nature," we shall not, with our imperfect knowledge, pretend to say whether it is to a man's happiness to live a long or a short time. We were made to die as much as

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to be born; and it is misleading to talk of any "law" or "tendency" which makes people get ill when they are well, or well when they are ill.

When compensation has been effected, then we do well to leave it alone. The patient is not suffering; we know he has a disease which may some time, and very likely will, bring about his death, but the difficulty being adjusted, it would be a very rash hand which would intrude; it would be meddlesome medicine which tried to improve what is well. But when the compensation begins to break down, we must begin to help it. Even then, in cases of aortic (sigmoid) disease, we should be very careful before we use strong drugs or active treatment. Many a life has been shortened by giving digitalis as a routine prescription in such cases. Our first object in such a case is to get the patient, if possible, to live a quiet and reasonable life. Secondly, to persuade him to adopt a proper regimen—somewhat scanty, dry food, with a little stimulant, for most of these patients are better for wine at their meals. To abstain from large draughts of water at meals is also important. Everything should be done to secure a daily evacuation of the bowels, to prevent flatulence, and bring about easy digestion. When, however, such an aortic case has gone on to the last stage, when there is dropsy and a weak, irregular pulse, with other symptoms more like those of mitral than of aortic disease, then we should treat it as a mitral case, and shall find that digitalis is very useful. Just so in the last stage of chronic Bright's disease, when the left ventricle is dilated, when dropsy sets in of a "cardiac" kind, then we may with great benefit treat it by digitalis.

Then, taking the other group of cases, a much commoner form, those cases which are mitral from the beginning, there we have a state which, incurable in one sense as it is, is, I think, more amenable to drugs, apart from other treatment, than almost any. You all must have seen what I can recall again and again—the water-logged patient, with legs enormously swollen, a full belly, and perhaps an œdematous hand, a jaundiced colour, a pale face, weak and thready pulse, unable to lie down, and if he drops asleep from fatigue, starting up in misery from short breath. We know his lamentable condition, and how very near he is to death. And you know how, by our treatment, we can, again and again, rescue such a patient most decisively and marvellously. First of all, get him out of bed; let him sit in an arm-chair by the fire-side, and he will often sleep and feel more rested than he has been for weeks. His legs swell to bursting, and it is well that they should, because the water which distends his legs, though it frightens him, is far better there than round his heart, and in his chest and abdomen. We tap his legs with a needle, or a small trocar and cannula, and as the water drains away the patient revives. Then we give him purges, and above all we give him diuretics, that wonderful pill which, I believe, was first prescribed by Mathew Bailie, of St. George's Hospital, which Addison called "the pill," made of digitalis, squill and mercury, with henbane. Such treatment will, again and again, restore a patient, as I am sure you have all seen, from a condition of misery and distress to one of comparative health.

May I refer to one or two cases illustrative of favourable prognosis, even in bad cases of heart disease? There was once a cabman who came under my care in hospital with all the symptoms of advanced mitral incompetence. I treated him in the routine way which I have mentioned, and he went out feeling perfectly well and returned to his cab-driving. Four times did that man come under my care in the same ward, and four times he went out free from every symptom—no dropsy, no jaundice, no dyspnœa, no discomfort; able to do his work again, and unluckily, also, able to eat and drink very much as before. It was only when he came in the fifth time that he died. We found *post mortem* a very badly damaged mitral valve, dilated right side of the heart, and all the other symptoms with which we are so familiar.

The next I will mention is an aortic case, that of an old gentleman who did not come under my notice until he was considerably over sixty—I think about sixty-seven years of age. He had a water-hammer pulse, a to-and-fro bruit, and symptoms of corresponding hypertrophy of the left ventricle. He was a remarkably intelligent man. I put the matter clearly before him, and particularly insisted on his never hurrying. He lived at Brighton, and on one occasion when he came to see me I was able to congratulate him on his good health. I said, "Have you followed my directions?" "Yes," he said; "last time I saw you I drove down to Victoria Station, and I was not sure whether I could catch a particular train. I said to a porter,

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'Has the train gone yet?' He said, 'No, sir; hurry up and you will catch it.' But I did not hurry up; I walked to the barrier and it was shut in my face. I bought a book at the stall and sat and quietly read it for one and a half hours, until there was another train, and went to Brighton quite happily." I said, "You deserve to live!" And so he did, for he reached the age of eighty, and then died of another disease.

Again, the late Sir Stafford Northcote, when he was an undergraduate at Cambridge, had rheumatic fever and a severe affection of his aortic valves. He was told that his life would be very short—it was in the early days of auscultation—and a physician when listening to his heart said, somewhat inconsiderately, "Sir, I hear your death-knell." But the patient survived to outlive his physician. Sir Stafford Northcote was doomed; he was told he could not live more than two or three years, and that he had better go to South Europe, to spend his time with the greatest comfort and pleasure he could. He did not take that advice, but went into the House of Commons, and became Chancellor of the Exchequer and Leader of the House of Commons. He lived to a good old age, and at last died from failure of compensation suddenly, painlessly and happily, having lived a useful and honourable career with those leaking valves all his life.

Even congenital cardiac disease is not invariably fatal at or soon after puberty. I have known two cases in which such a patient lived to be fifty, and may have lived longer.

Such cases show us how we are justified in not despairing even where there is disease of the heart of that kind which we are obliged to confess is incurable, and which we cannot alter because the mischief is done. Even in the worst cases, even where the valvular lesion is due not to rheumatism but to septic infection, we shall not altogether lose hope. I once had a patient who was a striking example of this. He had all the symptoms of ulcerative infective endocarditis, and we watched two aneurysms form in him, one in his radial artery and one in his *dorsalis-pedis*, as the septic emboli were carried from one part to another. But that patient recovered, and walked out of the ward to resume his work. One may say that such cases are "cured" as much and in the same sense that a broken bone is cured by the surgeon, by putting it into favourable conditions to bring about repair. These are some of the favourable cases I have met with, and I doubt not that some of my hearers have been so happy as to see the like.

CLINICAL LECTURES

ON

DISEASES OF CHILDREN.

LECTURE II.

THE ARTIFICIAL FEEDING OF INFANTS.

Delivered at the London Hospital Medical School, November 8th, 1902.

By ROBERT HUTCHISON, M.D.Ed., M.R.C.P.,
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GENTLEMEN,—We approach to-day a subject which exceeds, perhaps, in importance any to which I shall have occasion to direct your attention throughout this course of lectures; I mean the subject of infant feeding. When you go into practice the question, "How is one to feed this baby?" is one which will occur to you with perplexing frequency; and although it is a subject upon which a great deal has been written, I think one may say this, that the more you read about it the more confused you are likely to get, for, it must be confessed, much that has been written on the subject serves only to darken counsel, and you will find it of very little use to you in practice. I shall not deal in this lecture with all the conceivable methods by which children might be or have been fed; I want rather to describe quite plainly the methods which I have tried myself, and which I think you will find most easy and simple to carry out in your ordinary work.

Now the first question you have to ask yourself is—why should artificial feeding of children be necessary at all? You know that in the natural state of things every woman is the source of nourishment for her own child, but you will not have been long in practice before you realise that under modern conditions it is the minority of women who can nurse, or do nurse, their own children. It would be unjust to the female sex generally to state that this inability to nurse their children is their own fault. I do not think that is true; I think, in the majority of cases, one can say that the woman is unable—not unwilling—to nurse her own child. Now, that is a phenomenon of very considerable gravity, but it is one which is not confined to this country; it has been found to hold good of the whole of Europe, and in America

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also.* It seems to be part of the price which one pays for civilisation. It would appear that under civilised conditions of life the tendency of women to nurse their own children becomes less and less; and I need hardly say that that is a social event of very great seriousness, because the physical advantages to the baby of being breast-fed, not to speak of the moral advantage to the mother of suckling, cannot be over-estimated. This is one, indeed, of the signs of physical degeneracy which makes some observers afraid that there may arise a barbarian and more virile race which will sweep away our western civilisation altogether. Be that as it may, you will see from what I have said how important it is that you should understand how best to make good the absence of the mother's milk, and how it becomes a matter of pressing importance in practice.

If a woman is unable to nurse her own child, obviously the next best substitute is to get another woman to nurse it for her; in other words, a wet nurse is the best substitute for the mother. But I do not think you will have occasion to employ a wet nurse often in practice. There are many practical disadvantages entailed in that course, and, as a matter of fact, *artificial* feeding is synonymous in most cases with *bottle* feeding, so that what we have to consider to-day is how best you can feed a child by the bottle. There is still another question which has to be disposed of before we come to the consideration of the best way to carry out bottle feeding, and that is, supposing a woman starts to nurse her own child, how are you to know whether the process is successful or not? In other words, what is to justify you in adopting artificial feeding in the case of a woman who has *begun* to nurse her own infant? Gentlemen, I know only one condition on the part of the child that justifies premature weaning, and that is *persistent loss of weight.* If a child is persistently losing weight when fed on the mother's breast, that is an indication, and an imperative one, for weaning, and there is no other imperative indication that I know of. Such a state of things is illustrated in this chart. It is that of a child seven weeks old which was being fed on the breast, and as we weighed it from week to week we found it increased in weight very slowly, or hardly at all. And as there was no digestive disturbance we concluded that the mother's milk was insufficient; and we accordingly weaned the child, with the result you see—namely, that the weight shot up enormously, and the child gained more in one week of bottle feeding than it had gained in two or three weeks of breast feeding. There is one caution I should give you here. Before you conclude that the breast milk is insufficient, make quite certain that you have corrected any digestive disturbance in the child; and you will find particularly that you have to correct, in breast-fed babies, constipation. This, as I shall have to point out later on, is the vice of breast-fed babies, as diarrhœa is the vice of bottle-fed babies. So you see the statement I made that progressive loss of weight on the part of the child is the only indication which justifies premature weaning, has to be qualified by this, that it only holds good in the absence of digestive disturbance.

Having disposed of these preliminaries, we have next to consider what substitute we can use for human milk. Now, in practice cow's milk will always be your substitute. I shall not consider in these lectures the merits of other forms of milk. Many other kinds have been recommended, but the practical conveniences of cow's milk are so great that one rarely uses any other. I cannot too strongly impress upon you, gentlemen, the fact that it is the casein which is the source of difficulty in the artificial feeding of children with cow's milk. There are chemical reasons, which I shall not go into now, why the casein of cow's milk is less digestible than the casein of human milk; but whatever those reasons may be, it is a fact which you will meet in practice that you have difficulty in getting infants to digest cow's casein. For that reason you must give the cow's milk in a diluted form. How much, then, should you dilute the milk? It is best to begin first of all, as a simple practical rule, with a dilution of half and half; that is to say, give as much cow's milk as water. If you like, you can use barley water or lime water as the diluent; these make it more easily digestible than plain water. If you compare a mixture so prepared with human milk you find that from a chemical point of view it works out as follows:—In human milk there is $1\frac{1}{3}$ per cent. of proteid, $6\frac{1}{2}$ per cent. of sugar, and $3\frac{1}{2}$ per cent. of fat. In cow's milk there is 3 per cent. of proteid,

* See Bunge, 'Die Zunehmende Unfähigkeit der Frauen ihre Kinder zu Stillen.'

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4½ per cent. of sugar, and 4 per cent. of fat. If you dilute with equal parts of water you have 1½ per cent. of proteid, 2¼ per cent. of sugar, and 2 per cent. of fat. This makes the proportion of proteid, roughly speaking, correct, but the sugar and the fat are too poor, and you must therefore make up the deficiency of these constituents. That you can do quite simply by adding two teaspoonfuls of cream and two teaspoonfuls of white sugar to every eight ounces of the mixture. The ordinary feed for a young infant is about four ounces, or a little over. So, in order to feed a child artificially, dilute the cow's milk with equal parts of water, and add one teaspoonful of cream and one teaspoonful of sugar to each bottleful of food; that is, assuming the cream to contain the average of fat of London cream, which is about 40 per cent. That gives you a mixture which quantitatively is a very fair approximation to that of mother's milk. Qualitatively, of course, it is not the same thing as human milk, and it never can be.

The next question is—how much of the mixture shall you give, and how often? I have put into your hands a table showing you the

Time Table for Infant Feeding.

Age.	Intervals by day.	Night feeds.	Quantity at each feed.
1 week . . .	2 hours	2	1 —1½ ozs.
2—3 weeks . .	2 ,,	2	1½—3 ,,
4—5 ,, . .	2 ,,	1	2½—3½ ,,
6—12 ,, . .	2½ ,,	1	3 —4½ ,,
3—5 months . .	3 ,,	1	4 —5½ ,,
5—9 ,, . .	3 ,,	—	5½—7 ,,
9—12 ,, . .	3½ ,,	—	7½—9 ,,

quantities which the child's stomach will accommodate and the proper intervals of feeding. You must not suppose that the quantities there given should be quite hard and fast; you must leave it to some extent to the baby's appetite. The best plan is to tell the mother to give the child as much as it will take at each feed.

The next point is—what sort of bottle should you use? The only sort of bottle to be recommended is a boat-shaped one with a rubber teat attached. The rubber-tube bottles so much used probably destroy more infant life than any other single agency, and it would be a good thing if their use was illegal here, just as it is in France and in many States of America. Impress upon the mother that the bottle must be kept very clean. With regard to the teat, many cases of failure are due to its having too small an aperture in the top. You may want to know when the aperture is big enough. It is big enough if, when you hold the bottle upside down, the milk drops slowly, at the rate of about one drop per second. It ought not to require a great deal of sucking on the part of the baby, or it will be apt to give the child flatulence, or the infant will leave off sucking before it is satisfied, and so cease to gain weight. There is a teat in use called the leech-bite teat, so named because the apertures are triangular in shape, like the shape of a leech-bite, which seems particularly liable to produce these difficulties.

The next point is—should the milk be boiled, or should it be given raw? That, gentlemen, is a point which has probably been more discussed than its importance really justifies. I advise you to give it neither boiled nor raw, but scalded. That may appear to you to be a distinction without a difference, but by scalding I mean raising the milk to the boiling point, or just short of that, and then taking it off the fire. The drawback to boiling the milk is, that there is a very slight risk of producing scurvy by it. But that can be easily remedied by adding fruit juice to the diet. I think the advocates of boiling milk have exaggerated the dangers of bacterial infection. Certainly tuberculosis is not *often* conveyed through milk. When we come to speak of tuberculosis I shall point out that in the majority of the cases children are infected *viâ* the bronchial glands, and that dust is the great source of infection for children, as it is for grown-up people, and the tendency to regard milk as the main source of infection is erroneous. In hot weather it is much more necessary to scald the milk, because thereby one diminishes the tendency to its going sour and causing diarrhœa.

Having given the food by the bottle I have described, and at the intervals and in the proportions pointed out, you have to ask yourself—how are you to know whether the results are satisfactory? Here, again, the only test that this mixture is

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suitable is the balance. At what rate, then, should the child gain weight? This is not difficult to remember, because a healthy child should gain almost one ounce a day, *i. e.* it should gain six ounces a week. Obviously, one of two things will happen on this mixture; either the child goes on gaining weight, or it suffers from digestive disturbance, colic, possibly diarrhœa, possibly vomiting. If the child does not gain weight, or if it suffers from digestive disturbance, what are you to do? I advise you to remember the possibility, particularly if you are dealing with a very young child, that this mixture is too strong, and that it may be advisable to make it more dilute. In this chart I show you an instance of such a result. It is the chart of a child eleven weeks old who was fed on milk and water, equal parts of each, which resulted in the production of diarrhœa and vomiting, and only a slight increase in weight. Then we gave one part of milk and two parts of water, and immediately the weight shot up and everything went well. This shows that it is possible for a child to lose weight because it is getting too much to eat. Very often you will find that in young infants you may require to begin with one part of milk to two parts of water instead of equal parts of each.

Supposing that even in this very dilute form the milk still disagrees, what are you to do then? I do not think you are likely to effect much good by diluting cow's milk to a greater degree than this. One does not often see children do well on a mixture which is weaker than one in three. Therefore, what you have to do is to look out for a more digestible form of milk than ordinary cow's milk. You will find that more digestible form in *condensed milk*. If, then, you fail to improve the child's weight by giving it a food consisting of one part of cow's milk to two of water, I recommend you to try condensed milk. I think some consider it a heresy to recommend the feeding of infants with condensed milk at all. That is not a just position to take up. Condensed milk is an extremely useful temporary expedient; if used with suitable precautions, particularly if the deficiency of fat is rectified, it may be quite successfully given for the first two or three, or even more months, of the child's life. It is quite true that the continued use of condensed milk is almost certain to result in the production of rickets if kept up until the child is six months old. But fortunately by six months the child's digestion has generally become capable of dealing with ordinary cow's milk. Well, what brand of condensed milk should you use? Never select skimmed or separated condensed milk at all. The brand selected should contain all the original cream.

There are two classes of such condensed milk, those which are sweetened and those which are unsweetened. I used to recommend unsweetened, because in theory that is the best form, but I have given it up because it is so difficult to obtain. In practice I always recommend Nestlé's milk; it contains 13 per cent. of fat, that is to say, all the original fat of the milk, besides considerable quantities of added sugar. The dilution I recommend to start with is one teaspoonful of condensed milk to six tablespoonfuls of water, not barley water, but ordinary water which has been boiled and then allowed to cool. I quite admit that, theoretically, from a chemical point of view, that mixture is lamentably insufficient, but I only know that if you take the trouble to add fat in some other shape to the diet you can get satisfactory results with it for a time. You can give fat in the form of cream, adding a teaspoonful to each bottle of food; or, as often happens in hospital practice, if one cannot get cream one gives cod-liver oil, using it simply as a food, for cod-liver oil emulsion and cream are about of equal value in fat. I have here charts which show how successful feeding may be with condensed milk. Here is one of a child which was given one and a half teaspoonfuls of condensed milk to six tablespoonfuls of water, and you see how steeply the weight curve rises.

You may ask—why is condensed milk so much more easily digested than ordinary cow's milk? I think it is largely because one gives it so dilute. This dilution of one in six means that the casein will be much diminished, and if you add acetic acid to such a mixture and compare it with peptonised milk you find you get about an equal precipitate in each case.

Supposing even condensed milk fails, what are you to try next? You may then fall back upon *peptonised milk*, although it is not much more digestible than condensed milk unless it is very carefully prepared. I recommend you to use for making it Fairchild's peptogenic powders. Each packet contains full directions, and satisfactory

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results are certainly obtained by their use. But if you give peptonised milk alone it may still be too strong, and in that case you must dilute it with one third or even one half of lime water. This will ensure easy digestion. So you see one may regard diluted cow's milk, condensed milk, and peptonised milk as the positive, comparative, and superlative of digestibility; and it is well to have in your minds some such scale of digestibility as that, so that you can adjust the diet for any particular child to its digestive power. If its digestion is very feeble you give peptonised milk; if it is somewhat stronger you give condensed milk; and if it is still stronger you give cow's milk. There are many charts on the wall which show the results of successful feeding with peptonised milk. This, for instance, was the case of a child which had colic and constipation when it was fed on ordinary milk. Then we put it on to peptonised milk and water, about half of each, and you see its weight rose very rapidly. Some people raise an objection to peptonised milk; they say you will so demoralise the child's stomach by that means that it will be unable to digest ordinary milk later on. I believe, gentlemen, that this is a purely imaginary objection. I have fed children upon peptonised milk for many months, but I have never had any difficulty in getting them to digest ordinary cow's milk later on. So I think you may dismiss this bogey from your minds.

But if you fail even with peptonised milk, what then? Well, gentlemen, the resources of civilisation are not yet exhausted; there are still some things you can do, and I should recommend you in a case like that, when you have tried peptonised milk and the child still does not gain weight, to try the effect of a little grey powder. I do not know how the grey powder acts in those cases, but I am satisfied that in many cases, even although there is no reason to suppose there is a congenital syphilitic taint, the administration of grey powder will in some mysterious way make all the difference to the child's digestive power, and I have charts which illustrate that fact. This is the chart of a case in which we had tried the positive, comparative, and superlative of digestibility, and still we got no satisfactory increase in weight. Then we put the child on to grey powder, half a grain twice a day, and immediately the weight went up. Grey powder seems to fatten some children, just as cod-liver oil does. Another child was fed upon milk and barley water in equal parts, but it was not doing well. Grey powder was then given, and you see how great improvement resulted, although in that case also a history of syphilis was extremely doubtful.

If, in spite of all you do, the digestion does not improve, you are justified in thinking you have to deal with a child who is incapable of digesting cow's casein. There are some such children, those who cannot digest the casein no matter how you present it to them. And you have in such cases to take away the casein altogether, and to do that the simplest way is to prepare whey from the milk. Curdle the milk with rennet and allow it to set into a solid mass; then stir it up and strain it through muslin. By that means you have freed the milk from the casein. Whey is insufficient nourishment alone, because it contains no fat, so you have to supplement the fat. The best way to do that is to add one part of cream to four or eight of whey. Instead of using cream to supplement the deficiencies of whey you may sometimes use one of the patent foods. I have used Mellin's food and whey successfully. Recently I had a case under my care in which I ultimately succeeded with Savory and Moore's food made up with whey when everything else failed; but usually whey and cream will answer best. By such means as this you will in most cases be able to tide the child over its trouble even if it be of very feeble digestive capacity, until such time as it has cut some teeth. When the child reaches its first dentition often a complete change comes over the digestion, and instantly its nutrition improves. It would seem as if some of the digestive glands only attain their full functional power when the first teeth are cut.

You must not be surprised or blame yourselves, gentlemen, if you find every now and then that there are cases in which you cannot find anything on which the child will thrive. There will be a residuum of cases of this kind, which will be smaller the more skilled you are in your feeding. But there *is* a residuum with whom nothing succeeds. These are cases which I shall have to speak of when I come to talk about marasmus; they go progressively down hill, and they die without one being able to find out what was the cause of the fatal result. Here I show you a chart

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which illustrates the course of such a case, in which we tried milk and water in equal parts; we tried it stronger, and it produced diarrhœa; we tried peptonised milk for a time, and it was succeeded by diarrhœa and vomiting, and then the child died. Nothing which you can find after death will account for this congenital feebleness of digestive power.

I now want to impress upon you one further point about artificial feeding of children, and that is that you should not lightly change from one kind of food to another. You will constantly be having children brought to you whose mothers say they have tried everything. In such a case you may take it generally that *nothing* has really been tried in the proper sense of the term. You cannot judge from one week's experience what any particular food is going to do for a child; you have to keep the child on a diet for a reasonable time, so as to give it a fair trial, and it is only if you are finding that it is not gaining weight that you are justified in concluding that the food it is having is not suitable for it.

Another question is—what should be your attitude towards the patent foods which are so much in use for infant feeding? I have put into your hands a sheet which gives the composition of those foods, from which you will see that many of them are entirely unsuited to be a complete substitute for human milk. I have divided them, for convenience, into three groups: First, one which is intended to be a complete substitute for human milk; secondly, a group which is intended to be made up with cow's milk, and in which the artificial part of the food is predigested; the third class includes those which contain unaltered starch. My advice to you would be to rule out the third group at once for all children under six months old. As regards the first group, they really present very little advantage over condensed milk, and I hardly ever have recourse to them. I admit that some children may be reared successfully on certain patent infant foods, but hardly ever if condensed milk and other methods of feeding have been properly tried and have failed. They all incur the risk of producing scurvy. Therefore, if you use those foods it is well to see that some fresh elements are introduced into the diet, such as grape or orange juice.

The second group of foods is sometimes useful for children who are getting towards the period of first dentition and who are not thriving very well because it is difficult to get them to take sufficient cow's milk. In these cases it is advisable to supplement the milk with some other food, and a dextrinised food is best. There is one class of those dextrinised foods in which the change in the starch takes place during mixing. I think it is more convenient to use a food in which the change has already taken place; and so, if one has occasion to use this group at all, one selects Mellin's food or Hovis food as being already dextrinised in preference to the others. But remember they are all deficient in fat, and their continued use is apt to result in rickets. Therefore, if you use them at all it should be with that danger clearly before your minds, and they should be given only for a limited time.

One or two other questions I must discuss briefly. If a child has been on the breast and you have not had recourse to any artificial method, and everything is going on well, when are you to wean? I want you to have clear ideas on this matter. It used to be said by many people that a child should not be weaned until he has cut some of his teeth. I think even Trousseau went to that length; he was guided in his weaning of children by the dentition. But you must remember always that a child may fail to have its teeth because it is rickety from being fed on the breast too long; therefore you see the appearance of the teeth alone is no sufficient criterion of when you are to wean. You may lay it down as a general rule that the child should be weaned between the tenth and the twelfth month. Mothers, especially in the lower classes, often keep on suckling their children, partly from motives of economy and partly to prevent another pregnancy, for too long a time. I have even known children fed mainly on the breast until they were two years old, and without obvious harm. But I think in such a case there is very likely to be detriment to the *mother's* health. Between the tenth and twelfth month is the rule which you should adopt for yourselves.

Another piece of advice which I should like to give you in this matter is, never to wean a child in hot weather if you can avoid it. The reason for this is that children who are weaned in hot weather are very apt to become the subjects of

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COMPOSITION OF INFANT FOODS.

Food.	Water.	Proteid.	Fat.	Carbohydrate.	Mineral Matter.	General Description and Remarks.
Dried Human Milk	—	12·2	26·4	52·4	2·1	**The standard of composition to which artificial substances should conform.**
GROUP I.						
Allenbury No. 1 (for children below three months)	5·7	9·5	11·0	70·0	3·75	Desiccated cow's milk from which the excess of casein has been removed, and a certain proportion of soluble vegetable albumen, milk, sugar, and cream added. No starch present. ½ oz. in 3 ozs. of water for a child of three months.
Allenbury No. 2 (for children of from three to six months)	3·9	9·2	8·2	75·2	3·50	Resembles the above, but contains some malted flour in addition. No starch present. 1 oz. in 6 ozs. of water for a child of six months.
Horlick's malted milk	3·7	13·8	3·0	76 8	2·70	A mixture of desiccated milk (50 per cent.), wheat flour (26½ per cent.), barley malt (23 per cent.), and bicarbonate of soda (¾ per cent.). Contains no unaltered starch when mixed. 3 teaspoonfuls (equals 22 grams) in 4 ozs. of water for a child of three months.
Carnrick's Soluble Food	5·5	13·6	2·5	76·2	2·20	A mixture of desiccated milk (37½ per cent.), malted wheat flour (37½ per cent.), and milk sugar (25 per cent.). When prepared according to directions the casein is partially digested, but a considerable amount of unchanged starch is left. 1 part to be mixed with 9 of water and boiled for a few minutes.
Nestlé's Milk Food	5·5	11·0	4·8	77·4	1·30	A mixture of desiccated Swiss milk, baked wheat flour, and cane sugar (30 per cent.). More than a third of the total amount of carbohydrate is in the form of starch. 1 oz. to be mixed with 5 ozs. of water.
Manhu Infant Food	8·8	8·13	5·6	76·5	1·00	A mixture of desiccated milk and malted cereals. When prepared according to directions contains a good deal of unaltered starch. A dessert-spoonful (equals 13 grams) to be mixed with 2½ ozs. of water.
GROUP II.						
Class A.						
Mellin's Food	6·3	7·9	trace	82·0	3·80	A completely malted food. All the carbohydrate in a soluble form. May be regarded as a desiccated malt extract. ½ a tablespoonful (about 5 grams), ¼ pint milk, and ¼ pint water for a child under three months.
Class B.						
Savory and Moore's Food	4·5	10·3	1·4	83·2	0·60	Composed of wheat flour with the addition of malt. When prepared according to the directions, most, but not all, of the starch is converted into soluble forms (chiefly dextrins). 1 or 2 tablespoonfuls (equals 1—2 ozs.) to be mixed with 2 or 3 tablespoonfuls of cold milk or milk and water, and ½ pint of boiling milk, or milk and water, added.

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Benger's Food	8·3	10·2	1·2	79 5	0·80	A mixture of wheat flour and pancreatic extract. When prepared according to directions, most, but not all, of the starch is converted into soluble forms. The proteid is also partially digested as well as that of the milk used in mixing it. 1 tablespoonful (about an ounce) and 4 of cold milk, then add ½ pint of boiling milk and water; set aside in a warm place for fifteen minutes, then bring to the boil.
Allenbury Malted Food	6·5	9·2	1·0	82·8	0·50	A mixture of wheat flour and malt. When prepared according to the directions still contains some unaltered starch. Designed for children above six months. One tablespoonful (about an ounce), a teaspoonful of sugar, and 3 tablespoonfuls cold water; mix and add ½ pint boiling milk and water (equal parts).
Diastased Farina	8·3	7·6	1·3	81·7	1·10	A malted farinaceous food. When prepared according to the directions practically all the starch is converted into soluble forms. 1 oz. of food, ½ pint cold milk, and 2 ozs. water. Heat slowly till it boils; boil three minutes, and sweeten if desired.
Coombs' Malted Food	7·9	12·1	2·8	76·8	0·40	A malted farinaceous food. When prepared according to the directions still contains much unaltered starch.
Nutroa Food	6·8	13·9	10·3	68·0	1·00	A mixture of cereals with the addition of a certain proportion of pea-nut flour, from which the somewhat bitter taste of the food and its high proportion of fat are derived. It is a self-digesting food, but when prepared according to the directions only part of the starch is converted. 1 oz. of the food to be mixed with 1 oz. of cold water, and ½ pint boiling milk and water (equal parts) to be added.
GROUP III.						
Ridge's Food	7·9	8·7	1·0	81·7	0·70	A baked flour, containing only 3 per cent. of soluble carbohydrates, the remainder being starch. Recommended to be made with milk *or* water. Made with water alone is a totally insufficient food.
Neave's Food	6·5	10·0	1·0	80·9	1·60	Resembles the above, but recommended to be made with milk and water.
Frame Food Diet	5·0	12·9	1·2	79·9	1·00	A thoroughly baked flour to which has been added cane sugar and some extract of bran. It is *not* specially rich in mineral ingredients, but nitrogenous matters are abundant, and contains much unaltered starch. ½ ounce to be mixed with a breakfastcupful of milk and water (1 of milk to 2 of water).
Opmus Food	10·9	9·1	1·0	78·6	0·40	A granulated wheat food. 1 teaspoonful to ½ pint of milk. Starch unaltered.
"Falona"	7·0	9·7	3·5	78·6	1·20	A mixture of cereals (oats, barley, and wheat) with a ground fat-containing bean. The food is thoroughly baked, but contains a considerable proportion of unaltered starch. A teaspoonful to ½ pint of boiling milk *or* water, or ½ milk and ½ water.
Robinson's Groats	10·4	11·3	1·6	75·0	1·70	Ground oats from which the husk has been removed. Rich in proteid and mineral matter.
Chapman's Whole Flour	8·4	9·0	2·0	79·7	0·90	A finely ground whole wheat flour. Not much superior in nutritive value to ordinary "households" flour. Starch entirely unaltered.
Scott's Oat Flour	5·8	9·7	1·8	81·4	1·30	A fine oat flour. Somewhat inferior in nutritive value to "groats." Starch unaltered.

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epidemic diarrhœa, and to be carried off by it. Therefore it is better to allow the child to be nursed a little too long than to wean it at a time when diarrhœa is prevalent.

A kindred question to weaning is—when should you add starch to the diet? When is it safe to begin to allow a child to have other food than milk? Experience shows one that the individual power of digesting starch varies enormously in different cases. I have known children who got starchy food from their earliest days, and yet, apparently, did not suffer from it. I recall, for instance, the case of twins being brought to me who had been fed with a strong decoction of barley water *without any milk* for three months, and yet they were not so thin as you would suppose, and had no digestive disturbance. I have also known a child fed upon Robb's biscuits, which contain a good deal of starch, without any harm, when one month old. On the other hand, one sees children in whom starch appears to produce fermentation, acidity, diarrhœa, and colic. So your general rule should be not to allow a starchy food in any shape until the child has cut a tooth or two, and then you can begin to give such foods very gradually and cautiously. There are some children, however, in whom it may be worth while to run the risk of giving a little starch sooner than that. Children who are not getting on well with milk, and who have a difficulty in digesting casein, should have the benefit of the trial of a little starchy food. I once got a lesson in this matter from a mother whose child I was attending for difficult digestion. We were struggling on with peptonised milk and making no headway. One day, a week after I had seen the child, it was brought back weighing a pound more than it weighed at the last visit, and it transpired that the mother had, of her own accord, added a considerable quantity of cornflour to the diet, and with complete success. The starch-digesting capacity of that child was evidently in excess of its milk-digesting capacity, and in this case the experiment was justified by the result. Anyhow, it is an experiment which cannot do much harm, because so soon as you find it disagree you can stop the food.

As regards the diet of children after weaning, I cannot go into details now, but you will find full information on the subject in the appendix to the hospital 'Pharmacopœia.'

In my next lecture I purpose dealing with the disorders of digestion, which are apt to result from the neglect of the rules which I have just brought before you.

CANCER AND TUMOURS OF THE STOMACH. By DR. SAMUEL FENWICK and DR. SOLTAU FENWICK (J. and A. Churchill, 1902).

UNUSUAL interest attaches itself to this handsome volume, dealing with such a serious and important subject as cancer of the stomach, the joint production of Dr. Samuel Fenwick and his son, Dr. Soltau Fenwick, for the senior editor devoted, through a long professional life, particular attention to gastric maladies in general, and this will be his last contribution to medicine, for he died at a ripe age since the publication of this book.

The work, from the physician's and the morbid histologist's point of view, is admirably carried out. Clinical facts, microscopic research, and analysis of statistics are thoroughly and carefully laid before the reader; and the bibliography is rich in German references, so when the reader has traversed the excellently printed and abundantly illustrated pages, he will realise that he has read all that can be ascertained about this dreadful and alarmingly frequent disease, cancer of the stomach, by careful observation in the wards, *post-mortem* room, pathological museum, and the histological laboratory. The chapter on treatment seems to us to need the collaboration of a surgeon, for though it deals with the surgical methods of palliating this dreadful disease, it is far too short to be really useful.

The Parasyphilitic Affections; the Curability of Tabes and General Paralysis by Intense Mercurial Treatment.—Leredde declares that, after having cured a case of tabes with mercurial treatment, after thorough researches upon the parasyphilitic affections, and cases of recovery from tabes and general paralysis, he is absolutely convinced that in syphilitic patients these diseases are not only syphilitic in origin, but can also be cured by mercurial treatment if the mercury be pushed to very high doses. The unsuccessful cases are due to incorrect treatment, and to the existence of the irremediable degenerations produced in these conditions, as in all other affections of the nervous system. When the day, which is not far distant, arrives when the syphilitic nature of tabes and general paralysis will be admitted as a demonstrated truth, the histologists will have to describe the lesions of syphilis in a broader manner.—*Medical Record*, Jan. 17th, 1903.

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WITH SIR WILLIAM BENNETT
IN THE WARDS OF
ST. GEORGE'S HOSPITAL.

TUBERCULOUS DISEASE OF WRIST.

THIS is a good example of rather acute tuberculous disease of the carpus. The boy, who is 16 years old, was, so far as he can tell, well six months ago, when he had what was thought to be rheumatism in the wrist, which from that time till now has been painful, sometimes more so than at others. He has been under treatment at another institution, but as he was not content with what was being done there he came to St. George's for further advice. There are two or three points of interest about the case. First of all, on looking at the patient you will see that he is exactly the sort of subject who would be liable to tuberculous disease. He is a capital specimen of what used to be described in the old times, though you do not see much reference to it now, as the coarse scrofulous type of constitution. That is to say, he is inclined to be dark, with coarse dark eyebrows and hair, and a tendency to the prolongation of the hair growth down the back; his skin is a little rough to the touch, and his look is rather dull and heavy, although the eyes are bright—such was the coarse scrofulous type of person in whom tubercle was common, especially in the joints. This tuberculous disease of the carpus arises under two circumstances. It may commence in the carpus itself, or, as sometimes happens, it may commence outside the joints in a ganglion which may have formed at the back of the wrist. What the origin was in this case it is impossible now to say. It looks as if it is a case of general disease of the carpus commencing in the carpal articulations, probably in the synovial membrane. If we operate we shall almost certainly find that the bones are all in a condition of advanced caries. It is well to bear in mind that in all the compound joints composed

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of a number of bones like the carpus and tarsus, extensive disease of the synovial membrane is liable to be associated with disease of the bones which progresses very rapidly. This is due to the nature of the process and the resulting interference with the nutrition of the bones. The whole synovial membrane surrounding the bones being diseased acutely, nutrition is so interfered with that caries and necrosis follow, conditions which could not occur to the same extent in joints composed of the ends of two or more long bones; extensive tuberculous disease may occur around the ends of long bones without any great change in the bones at all, as the nutrition of the bone ends is not dependent on the synovial apparatus. It is worth bearing in mind that some of these cases of tubercle of the carpus and tarsus commence in a ganglion outside the joint, which becomes involved secondarily. This naturally only occurs in cases where the ganglion is in communication with the joint. You will find it is very commonly said that the ganglia about the wrist rarely, if ever, communicate with the wrist-joint. It may be less common for the ganglia to communicate with the joint than not, but in some cases the ganglia do communicate with the wrist. There is no precise sign which will tell you whether a ganglion does or does not communicate with the wrist, and there is no anatomical bursa normally communicating with the joint. There is, however, no doubt that ganglia occurring to the radial side of the middle line at the back of the wrist have a tendency to communicate with the wrist-joint—that is to say, either with the radio-carpal articulation or with the articulations of the carpus; those to the ulnar side, on the contrary, do not, so far as I have seen, ever communicate with these joints. This is a useful point to bear in mind, because it makes the ganglia on the thumb side of the middle line of more importance, from the point of view of secondary infection of the joint beneath, than those on the little finger side, in which secondary implication of the joint beneath rarely, if ever, happens. There is no difficulty about the diagnosis in the case before us. The whole of the carpal region is swollen, both on the dorsal and on the palmar aspect; you will notice how exactly the swelling follows the outline of the articulations. There is a little œdema extending from the swelling on to the back of the hand, exactly the sort of thing you would expect in such an acute case. On manipulation you find that, although gentle flexion or extension causes no pain, if the forearm bones be taken in one hand and the fingers in the other and the parts then pressed together, the patient will flinch, distinctly showing that pain is caused. You need not ask the patient if the process is painful; his face will indicate that. Pain produced in this way is certain evidence that the bones are diseased. There is no crepitation, as the bared cancellous surfaces of the bone are not in contact, because they are separated by granulation tissue. So we have here, then, a rather advanced condition, which is acute; and the question arises—what shall we do for it? The boy is sixteen years of age, and looks younger. So far as we know, he has no visceral complications, and the point arises whether the disease at the present moment is in a condition in which radical measures should be taken—that is to say, operative measures—or is the case one in which we should be justified in temporising, in the hope that we may get a complete cure spontaneously in the form of ankylosis? Before we come to the question of treatment, it may be well to consider for a moment whether in these cases there is any particular relation between diseases of joints of this kind and pulmonary tuberculosis. It has been held by some people, and with a certain amount of justice according to their statistics, that tuberculous disease affecting joints like the carpus, tarsus, and the elbow especially, for some reason which is not quite clear, is more prone to be associated with tuberculosis of the lung than are affections of the same sort in other joints. If that is true—and, as I have said, there seems to be some reason for believing that it is true, the question of treatment is, to some extent, conceived, because, if what I say is true, if the disease occurs in a joint in which the tendency to pulmonary complication is not so strong, we may be inclined to temporise and attempt to save the joint for a longer period than we should in a case like this, in which there is a stronger probability of further tuberculous mischief arising elsewhere.

With regard to the treatment, there are three courses open to us—(1) to temporise with a view to saving the wrist intact although ankylosed; (2) to perform excision of the joint, and (3) to amputate. In this boy I think it is practically

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certain, taking not only his aspect and general condition into consideration, but also the circumstances in which he is placed in life, that we shall not save the wrist intact; it is practically certain that an operation of some sort will have to be done. If the boy were in a different position in life there would have been no question of his limb being sacrificed, because it would not have been allowed to become so grievously diseased; at least, I cannot conceive that it would have been allowed to get into such a condition. Taking the case as it stands, there is clearly nothing to be done short of an operation. The question therefore resolves itself into this—shall we excise the wrist, or shall we amputate through the forearm? The results of excision of the wrist in suitable cases are admirable, and one would naturally hesitate to sacrifice the hand unless the circumstances of the case rendered any attempt to save it too dangerous to be justifiable—the main danger being a further dissemination of tubercle, the tendency to which would be very slight if the whole of the disease could be removed. Seeing the enormous value of preserving the hand, even if the wrist be stiff, in a boy like this, I should advise an attempt to save the hand by excision, although amputation is actually by far the safer treatment, and of course the quicker in result too. Amputation would of course follow if the progress of the case did not prove satisfactory. After the excision I should not use the usual antiseptic dressings, but should keep the part continually immersed in a bath, either of normal saline solution or of boric acid solution, the part having been gently fixed on a metal splint to secure the necessary rest. This method of after-treatment has, in my experience, been much more successful than any other in these advanced cases. The solutions are either changed four times in the twenty-four hours or are kept continually refreshed by properly balanced inflow and overflow. The method is most comfortable to the patient, since there is no changing of dressings, and most convenient to the surgeon, as the parts are open to inspection at any moment. The time during which the continuous bath treatment must be persisted in depends upon the nature of the case. In a case like this a month at least will probably be necessary. If the treatment can be managed as much as possible in the open air, so much the better. Such is the after-treatment which I strongly advise you to adopt if possible in any case of tubercle like this, in which there *is any doubt about the complete eradication of the disease* by the operation. If the eradication is *clearly complete*, the usual antiseptic or sterile dressings should be used.

Delayed Union of Fracture of Femur.

Here is a case of fracture of the femur in which nine weeks after the accident there is no union. The fracture was treated by splints in the ordinary way by my house surgeon. At the end of six weeks the splints were taken off and afterwards replaced, and you can see now how the case has gone on; there is really no union worthy of the name. In point of fact, the bones are quite ununited. It is, however, not right to call this an ununited fracture of the femur in the ordinary surgical sense, because the time since the accident is not long enough to justify our speaking of it as such. An ununited fracture means one which will not unite after all ordinary means have been taken to promote its union. This is at present merely a case of fracture of the femur in which, at the end of nine weeks after the ordinary treatment by splints, there is delayed union. Whether the case will finally unite or not remains to be seen. Now there is clearly some reason for this defect, and, as you know, there are many causes, local and otherwise, given for absence and delay of union after fracture. This man affords a good type of one of the constitutional reasons for non-union, and his case is one which you will do well to bear in mind. You see he has a very characteristic aspect; so much so is this the case, that when he came into the hospital I stated that a very slow union might be expected, and that non-union might even finally follow. The man is a painter by occupation; for many years he has had to deal with lead in various ways, and the constant working with lead seems to have the effect of interfering with union of bones after fracture. The patient has a peculiar pale aspect, very difficult to describe, but which you can generally see in people who have followed occupations like his for a long time. He is prematurely grey, and although he is only forty-five years of age, he looks very much older. His arteries are more rigid than they should be in a man of his age. This change towards

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senility, which takes place comparatively early in people of this occupation, seems to be associated with defective power of repair in fracture. That being the case, if we wish to alter this condition of things we naturally should, under ordinary circumstances, have to attack it from the constitutional point of view. But this man has been in these circumstances almost all his life, so it is very difficult to alter his constitution at this period. The only internal remedy likely to help you in such cases is iodide of potassium. It seems that this drug does at times help to some extent. But before administering any internal remedy you should ascertain with a good deal of care what the condition of the urine is; it may contain albumen or it may be strongly phosphatic. In this case it is normal, although perhaps rather more limpid than usual; the kidneys, therefore, may be considered to be normal. The question now is whether some immobile form of splint should be applied for a further period, or whether operative measures (*e. g.* wiring, pegging, etc.) should be resorted to. Under ordinary conditions I should recommend wiring in this case, though I am not a strong advocate of wiring indiscriminately in cases of fracture. I should do this because in a man of a different constitution to this the holding together of the two ends of bone by means of wires or screws would probably be followed by a more rapid result than we shall get now, and, virtually, without more risk. In this case I should not recommend wiring or screwing, because the interference with the bones by wiring, screwing, or pegging is not at all unlikely to be followed by necrosis in a lead-contaminated subject. There is something peculiar about these leaded patients in regard to their bony constitution. I do not know what it is exactly, but their bones do not stand interference well, and for that reason I put wiring out of court in this case, unless it becomes absolutely unavoidable. I shall therefore immobilise the fracture by means of a moulded leather splint, with side irons, and get the man up on crutches, instructing him to bear weight from time to time upon the damaged limb—this is an important point. In order to promote union in people of this sort a certain amount of irritation about the seat of fracture is desirable. There is a treatment as old as the hills, which has been recently discovered again, as so many other things have been in medicine and surgery, being now called the ambulatory treatment of fractures, which merely means that the fractured limb is put up in some form of splint which allows the patient to bear weight upon the limb—in fact, to walk upon it in a modified way. Of course, every time the foot is brought to the ground the bone ends come together and there is produced a rubbing or friction, which may bring about the necessary irritation for the production of callus. Under those circumstances the movement of the fracture which takes place whilst the patient is walking about, instead of preventing union, often produces callus and brings about union in cases in which it has been unnaturally delayed.

This case is a good one for observation for the following reasons:—First, that a broken bone in people who follow trades in which they dabble with lead continually is more serious than it is in subjects who are otherwise occupied. Secondly, that you should never fail to warn the patient in a case of this sort that he may be laid up longer than the ordinary run of people who have broken bones, for the reason that there is often delay in union, and that sometimes the bones finally refuse to unite. The present case may be one in which there will be no union. If that proves to be so we shall have to wire the fracture, and if this should be necessary we must be prepared for some trouble in connection with the treatment, and perhaps, also, some necrosis. Another point about the wiring of this class of case is this—and I wired one or two of these fractures before realising the importance of the matter—that the wires or screws appear to break away much more easily than in other cases. It is also said, and, I think, truly, that people of this type and calling are liable to fracture from slight causes.

A Case of Adenoma of Thyroid.

Let me call your attention to a few facts in connection with a case of adenoma of the thyroid upon which I operated a few days since. I had no opportunity of speaking of the case before operation, as the symptoms became urgent, necessitating immediate interference. The patient is in the Drummond Ward, and the thyroid tumour was very large, pushing the trachea greatly over to the right side. A doubt was expressed as to whether it was a cyst or not; it turned out to be a solid adenoma. There was very little difficulty in getting

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it away, and no hæmorrhage worth mentioning occurred, in spite of the large size of the tumour, which was enucleated. Enucleation of these tumours is not, so far as I have seen, accompanied by bleeding if the operation is properly done.

The first point of interest in the case was the extreme dyspnœa from which the patient suffered; this was so great that upon lying down she was in danger of asphyxiation. There was also a most characteristic difficulty in swallowing. The peculiarity of swallowing met with in bronchocele, which allows of solid food being taken, whilst liquids are rejected, is interesting, since it is exactly the reverse of what happens with patients suffering from obstruction in the gullet, in which fluids can generally be taken, whilst the swallowing of solids may be quite impossible. Since the operation in which the adenoma was enucleated, both these distressing symptoms have disappeared. As I mentioned to you the other day in connection with another thyroid case, the explanation of the dyspnœa in a case of that kind is simply that there is pressure on the trachea; it is not due to any secondary affection of nerves or anything of that kind—the cause is purely pressure. It is sometimes said that the pressure is due mainly to the weight of the tumour. The reason which is given for this statement is that no matter how bad the dyspnœa may be during the day when the patients are up and about, it is always worse when the recumbent position is assumed at night or at other times; whilst lying on the side it is not so bad. The chief factor, however, is not the weight of the tumour, but, as I have mentioned to you in speaking of another case, the unnatural tension of the cervical fascia. If you think of the situation of the thyroid gland and of its capsule, which is composed of strong layers of cervical fascia, you will see that, as the tumour in the thyroid grows, it must of necessity increase the tension of the cervical fascia; and so produce pressure on the trachea. It is that which is the main cause of the dyspnœa. To relieve the dyspnœa, your immediate object is to relieve the tension of the cervical fascia. In removing the tumour in this woman we relieved the tension in the cervical fascia, because we removed a large mass from between its layers. If you ask me why it is that the removal of these tumours so *instantly* relieves the difficulty of breathing which sometimes exists in these cases, I should say it is not so much the removal of the tumour itself as the slackening of the cervical fascia which follows that removal. In support of this view, it is, I suppose, well known that immediate relief to dyspnœa in thyroid enlargements can almost invariably be afforded by free division of the deep cervical fascia in the middle line.

A Case of External Urethrotomy.

There are a few points of interest in connection with this case. The patient is a man of middle age, who was admitted on account of an intractable stricture with perineal fistula. External urethrotomy was necessary, and it was done in the usual way, a full-sized catheter having been afterwards introduced and tied in. The progress of the case was excellent as usual. The catheter was allowed to remain *in situ* for eight or nine days, rather longer than, speaking generally, it is safe to leave a catheter in the bladder without changing the instrument. It is not wise, as a rule, to leave a catheter tied in the bladder for more than a week at the outside in cases where there has been vesical irritation and perhaps cystitis, because the tendency to phosphatic deposit on the end of the catheter is often very strong, which may for obvious reasons lead to awkward complications under certain circumstances. At the same time, it is wise in cases of this kind not to attempt to change the instrument sooner than it is absolutely necessary, because the longer an instrument in these cases is left *in situ* the more easy is it to substitute another instrument upon the withdrawal of that introduced at the time of the operation. Opinions, of course, differ as to the desirability of retaining catheters in the urethra in these cases; my experience is that a better result is obtained if a catheter can be kept in the bladder for three weeks at least after the operation. If this is done it entails a change of catheter at least twice, or perhaps thrice. The first change of the instrument may be the simplest matter possible; on the other hand, it may be extremely difficult, or indeed impossible, without damaging the parts at the seat of operation beyond what is justifiable. In other words, the first instrument may slip in without any trouble, or it may refuse to go at all. Whether it passes successfully or not will necessarily depend to a great degree upon the dexterity of the manipulator; but in the most dexterous hands diffi-

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culty arises sometimes. In this case it so happens that, although I made the first attempt at changing the instrument myself, great difficulty arose, and, although I told those of you who were present that I had no doubt I could if I persisted finally introduce the instrument, I did not think it right to persist too long in my attempt, because there was a danger of opening up afresh the parts about the urethrotomy which by this time were on a fair way to healing. I therefore left the case entirely alone, so far as the passage of any instrument was concerned, and I shall make no further attempt at passing an instrument until a month has elapsed after the operation. I may then be able to pass an instrument with moderate ease or I may not, but in any case I shall not persist in any attempt if blood began to show itself coming from the urethra after moderate manipulation, but shall postpone any further attempt for a couple of months or thereabouts, when a small wax bougie would undoubtedly pass and act as a guide for other instruments to follow. At first sight it may seem to you that the failure to introduce a catheter or bougie at the end of a fortnight means a failure of the urethrotomy operation, but this is not so, and you may be quite sure that well-considered delay in such a case will lead to a better result ultimately than indiscreet haste in the introduction of an instrument for academical reasons. The great point in the management of a case like this is to avoid unnecessary injury to the cut parts, by too persistent or forcible attempts to introduce an instrument. Although it is, of course, generally best that instruments should be methodically used after the operation, excellent results not infrequently follow in some of these cases in which it has been found impossible to pass instruments subsequently. I have often mentioned to you before that a very free stream of urine can sometimes be passed through a urethra down which the smallest catheter cannot be made to go; it does not, in fact, always follow after these urethral operations that because a catheter cannot be passed the stream of urine is small—a practical point worth bearing in mind, because it is foolish to worry a patient with futile efforts to introduce instruments if a stream of urine equal to a No. 7 or 8 English catheter can be passed naturally. Therefore, before you persist in attempts at passing instruments in any case of this kind, ascertain to what degree of freedom the patient can pass his urine. See him micturate, and note the size of the stream—never take a patient's word in estimating the calibre of the stream of urine; and if, as I have said, you find a stream of the size I have mentioned, whilst you are unable to pass a small instrument, leave the case alone *unless the stream is gradually decreasing in size.* This may seem to you perhaps very bad teaching, and not the course which you would be led to think is right by reading the ordinary books on the subject, but I am persuaded that it is the best practice. The two cardinal points to which I attach most importance in the management of stricture are these:—1. The avoidance of worrying the urethra with instruments more than is really necessary. 2. The avoidance of straining during micturition. If patients could only be made to understand the importance of avoiding the use of any force in expelling the urine, more than half the troubles caused by stricture would cease to occur.*

Topical Blood-letting as a Derivative and Revulsive Remedy in Congested and Inflamed Hæmorrhoidal Blood-vessels and in other Affections.—W. Bodenhamer refers to the older practices of venesection and arteriotomy, and deplores the fact that they have become so obsolete, for he does not believe that there are any other measures so valuable as these in pleurisy, pleuro-pneumonia, and sunstroke. Leeches applied to the ano-rectal region are beneficial not only in maladies of these parts, but also in chronic affections of the liver, spleen, colon, and contiguous organs. In hæmorrhoidal prolapse the leeches should be applied not directly to the parts, but at some distance. Similarly in certain affections of the feet it is better to abstract blood from the arm. In hæmorrhoidal cases the best region for application, in the author's opinion, is the lumbar. From five to fifteen leeches may be applied daily, according to the condition of the patient, until he is relieved. After the acute inflammation has been subdued, operative measures may be considered in those instances in which the tumours have become regularly organised.—*New York Med. Journ.*, Jan. 17th, 1903.

* See a lecture on "The Management of Stricture," by Sir William Bennett in the 'Practitioner' for June, 1900.

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A CLINICAL LECTURE

ON

SOME AFFECTIONS OF THE SKIN OF THE HANDS.

Delivered at St. Bartholomew's Hospital.

By J. A. ORMEROD, M.A., M.D., F.R.C.P.,

Assistant Physician and Physician-in-Charge of the Skin Department, St. Bartholomew's Hospital.

GENTLEMEN,—There are two parts of the body where disease of the skin is both common and important. One of these is the hands, and the other the face. The face, of course, is important, because people are very concerned about anything which affects it. They may have a rash over the whole body, and yet may not care anything about it, but when it reaches the face they will come to have it cured. Disease of the skin of the hands may interfere with the avocation of the working man, while to the leisured classes it is inconvenient, unsightly, and may prevent the customary washing. Exposure is one reason why the hands are so commonly affected; another is their relation to the circulation, standing as they do at one extremity of it; a third is their structure. The structure of the skin even of the different aspects of the hand varies. For instance, I hope to-day to show you some affections on the dorsum of the hands which would be very different from an affection on the palmar surface of the hands, no doubt because of the difference in structure of these parts.

Of scabies, which is one of the commonest diseases of the hands in hospital practice, you see so many examples in the Surgery that I shall pass over that subject and come to another common disease, viz. eczema of the hands. The characters of such an eczema vary a great deal, according as it is on the back of the fingers and hands, or on their palmar surface. This is to be accounted for by the difference of structure in these parts; for on the dorsal surface of the hands the epidermis is thin, and there are many hair-follicles; whereas on the palmar surface the epidermis is very thick, and there are no hair-follicles; while, on the other hand, there are many sweat-follicles on the palm. Eczema may occur as a papular or vesicular rash beginning be-between the fingers, and then it is very readily confounded with scabies. Like scabies, it itches much, but scabies is said to itch much more at night. Like scabies, too, it may affect the inter-digital clefts, but eczema differs from scabies first in the absence of "burrows;" and secondly, in that the vesicles are generally not so discrete or so irregularly scattered as in scabies, but are more often grouped into little patches. Again, while in eczema there may be discharge and crusting, there is not such distinct pustulation as in scabies. Eczema on the back of the hands and forearms may be due to very various causes. You know that eczema often has a double cause; there may be some sort of constitutional basis, and at the same time a local cause. In eczema of the back of the hands I think the local cause generally predominates. I have a case here which seems to show both a general and a local factor. This woman came with what is called a seborrhœic eczema. It began by scurfiness of the head, which spread down to the body in the form of scaly reddish plaques. She was given a soap and spirit wash for the head, and after that the hand became bad; and, in her opinion, the particular affection of the hands was determined by the soap and hot water which she used for washing her head. There is not very much eczema on her hands now. The results of it, however, are shown in the finger-nails, which are roughened, while some of them are thin and cracked. This is due, as most affections of the nails are, to an antecedent disease in the matrix of the nail, which in this case was an eczema. In many cases the nails are much more affected than they are in this woman; they may be greatly thickened, and piled up with dirty incrustation, a condition which may take longer to cure than the affection of the hands which has given rise to it. Such a thickened nail has to be destroyed by frequent application of salicylic acid, followed by paring and rubbing down; and we then trust to Nature to provide a healthier one, after the disease of the matrix has subsided. The local causes of eczema of the hands are many, as you know, for the hands are exposed to irritation from many sources—such as sun, wind, and cold; hot water, soap, and soda, and dirt in the case of scrubbers and washer-women; septic matter in the case of dustmen and sewermen; antiseptic but irritating chemicals, such as mercurial and carbolic lotions, as surgical

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dressers may have reason to know. Colour workers, who have to deal with aniline dyes, are subject to eczema of the hands. Printers and bootmakers and French polishers also frequently have it, the latter from the potassium bichromate which is used in the polish. Some special forms of wood dust appear to irritate the skin and produce eczema. Thus a carpenter in the skin department told us he had had eczema of the forearms when he worked with teak wood, but not with any other kind. There are many other causes, and you can find out a good deal by questioning people about the things which they use in their occupations, and whether their fellow-workers are affected in the same way. Most of these patients (except, perhaps, the washerwomen and charwomen) have it on the dorsum rather than on the palm of the hands. It is generally rather patchy than diffuse, or it may be even limited to the follicles, of which there are large numbers on the backs of the fingers and forearms; and once lodged in them it is difficult to cure. It may spread to other parts, such as the face and neck, being apparently carried thither by the fingers. I show you a patient who is a printer, and who for two or three years has had a rash on his arms, which I think we may reasonably ascribe to the ink which he uses in his trade. He says many of the men in his workplace have the same condition on the fingers and arms. But a second cause came in, in his opinion, when he was recently vaccinated; he thinks that after this the rash became very much worse, and that it assumed rather a different character. Certainly around the area of the vaccination marks there was a good deal of thick yellow crusting, like that of impetigo, and the hands and arms showed a tendency to become impetiginous too. I have no doubt he had originally an eczema, probably of the follicles chiefly, and it is not improbable that it became infected with cocci after the vaccination, and thus became worse. I have treated it as I should an impetigo, with ammoniated mercury and zinc. Of course the difficulty in his case is that he remains at work, and when a man remains exposed to the cause which produced the disease it is rather difficult to get him well. His eczema affected the fingers and the forearms, and not the palms of the hands at all, probably because the epidermis is thick there. The treatment of eczema on the back of the hands may be difficult. First of all you have to remove the cause, which you cannot always do. Secondly, washing with soap and water, which most people find necessary, may make the eczema worse. In the acute stages you must apply, as best you can, sedatives and astringents, lead or zinc lotions; the ordinary thick calamine lotion not only acts as an astringent, but forms a sort of protective crust. Small patches can be further protected by the application of Unna's paste or his zinc and ichthyol mulls, or to a certain extent by sticky ointments such as the ordinary zinc or ammoniated mercury ointments. Dr. Jameson, in his book, strongly recommends Lassar's paste for eczema of the back of the hand. This is made with starch and zinc oxide, two drachms of each with half an ounce of vaseline; it helps to dry up a moist eczema, and you can add to it a variety of useful medicaments, such as salicylic acid, resorcin, ichthyol, etc.

We now come to eczema of the palm; and this differs in character from eczema of the dorsum of the hand, because, instead of an eruption of vesicles or raw patches, or an irritable pustular folliculitis (all of which are common on the dorsum), its chief manifestation is the formation of thickened firm layers of epidermis, in which painful fissures and cracks may appear. It is then known as eczema rimosum. It is usually limited to the palms, but it may creep round to other parts, especially the tips of the fingers, where cracks are particularly inconvenient. It generally begins as a thickened patch in the centre of the palm, with a slight area of redness around, and this patch spreads until the whole palm becomes inelastic, and painful, especially when it is cracked. It is very tempting to ascribe this disease to local causes, such as washing and scrubbing, but that does not seem to cover the whole ground, because the soles of the feet are sometimes affected as well. This patient, for instance, works as a confectioner's assistant, and you might think that her occupation started her disease; but there must be some additional factor, because she has worked at that business for twenty-four years, and yet she has only lately developed eczema. Her whole hand is involved, though in most cases eczema of the palm remains limited to that part. Her nails are not much affected. There is no discharge of any kind. The hardness of the epidermis in these

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cases interferes with the success of the treatment. We endeavour to get rid of this thickened epidermis, and to restore the skin of the palm to a normal state of nutrition. Perhaps the best local application for this purpose is salicylic acid. To out-patients here I give it in the form of an ointment, twenty or thirty grains of salicylic acid in the compound ammoniated mercury ointment of our pharmacopœia. It may also be applied in the form of a "plaster mull." I show you one of these. It consists of a backing of coarse muslin, on which is spread a thin layer of gutta-percha, and on this the plaster, consisting of the drug mixed in with some adhesive substance. It is kept on continuously, but is removed once a day, the hand being then soaked in hot water, the epidermis scraped or rubbed down, and the plaster reapplied. But the ointment is simpler for out-patients to apply, and answers well.

What other affections are there which may be limited to the palms of the hands? There is one very important one, namely, syphilis. That disease may affect the palm, and it may look very much like an eczema in that situation. But, unlike an eczema, it is unilateral. It is also said to begin as one or two reddish infiltrated patches, which become scaly, and then spread till they form a patch with gyrate edges; while eczema usually begins as a single patch in the centre of the palms. I have no case of the kind to show you. But I have here an interesting case in a woman who has what I believe to be eczema, and yet at the same time has a syphilitic taint. She comes here for the eczema, which is now going on very well, and there is little to see of it. She had an eruption on the palms of her hands, and on one wrist a patch of redness which spread in a circular form. Such a gyrate rash raises in itself the suspicion of something syphilitic. Further evidence of syphilis is supplied by the numerous scars which you see on her face. She says hard lumps came there and left scars. No simple eczema leaves a scar. She has also a very characteristic tongue. So she has been treated partly locally, and partly by the usual constitutional measures. Simple psoriasis hardly ever affects the palms of the hands; I will not say never. Lichen planus may affect the palms, but usually there is a rash elsewhere. Curious as it may seem, some medicines may produce an affection of the epidermis of the palms, notably arsenic. You will find in the account of the Manchester epidemic of arsenical poisoning that thickening of the epidermis upon the palms and soles occurred in very many instances, and it is very useful to remember that when making the diagnosis of chronic arsenical poisoning. I remember a man, an out-patient at the Queen Square Hospital, who was having arsenic in conjunction with iodides in his medicine. He remarked that the skin of his hands had all "gone curious," and I imagined it might be the arsenic. I stopped the arsenic and the skin got right again; but after resuming the arsenic the same condition of the skin recurred. I have seldom had a more convincing proof of the effect arsenic may have on the skin than that.

The last patient whom I show to you is a man who suffered from an affection of the hand known as cheiropompholyx. Some people consider it to be the same as eczema, but I do not think that is the view usually adopted. It occurs in the form of vesicles and bullæ on the hands and feet, and it is preceded by itching and burning. The eruption consists of fairly large vesicles, which are generally compared to sago grains, and these seem rather deeply seated in the skin. They come out along the sides of the fingers and on the palm of the hand; sometimes on the dorsum; and sometimes also on the feet. They contain neutral or alkaline fluid, and they do not, it is said, burst of themselves, like the vesicles of eczema, and if they are broken they do not leave a raw discharging surface like eczema. They simply dry up, leaving large desquamating flecks of skin. This man came to the skin department three days ago with a most characteristic eruption, which you still can see. He has had something of the same sort on the feet. As to the nature of this eruption, it was originally described by Tilbury Fox under the name of dysidrosis, and it was thought to be due to dilatation of the sweat-follicles. Certainly it occurs mostly in the summer-time, and in people who sweat readily; but this man's case is exceptional, in that the rash has occurred in the winter. Unna thinks cheiropompholyx is an exudation in the neighbourhood of the sweat-glands rather than a dilatation of the sweat-glands themselves, and that it is caused by a bacillus, which ordinarily is latent in the skin, but which may be stirred into activity by the profuse sweating. This patient has had the

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same condition previously, and then it was in the summer. Recurrence is indeed one of the characteristics of cheiropompholyx. It generally occurs in people who are out of sorts and feeble. This young man does not look very robust, but otherwise I can find no cause for his disease. The rash runs its own course, dries up, and gets well. There is nothing to be done for it except to use protecting ointment. He is having boracic ointment, which he thinks is doing him good, and I shall give him a tonic to improve his general health.

There are other affections of the hands which you should remember, such as the erythemata, chilblains, lupus erythematosus, &c.; but I have no cases to show you at present, and no further time in which to speak of them.

Cinnamic Acid.—On the cinnamic-acid treatment of tuberculosis, Robinson says that to regard the drug as a specific is ridiculous, and it is not so claimed by Landerer or Cantrowitz, his most ardent follower, but it is probably a useful adjunct in the treatment of the first stage of tuberculosis. The doses must be small to begin with and be very gradually increased. Landerer insists on this point. It is contra-indicated in cases inclined to hæmorrhage, and soon after hæmorrhage, and when the temperature is above 101° F. Should a rise of temperature follow its injection the dose is too large, and the next dose should be diminished one fourth or a half. There is no justification for mistrusting Landerer's statements, but it must be remembered that similar results are obtained without the use of this drug. The treatment is harmless if directions are followed. Too large doses may cause excessive serous exudation around the tubercle foci, and carry some bacilli into the circulation. Given by the mouth the doses of sodium cinnamate may be large, but it remains to be shown whether, thus administered, it has any effect on the tuberculous processes. Taken altogether, Robinson's impression of sodium cinnamate as a curative agent in tuberculosis is a negative one. It is probably a useful stimulant, but he prefers creosote or some of its derivatives. —*Journ. Amer. Med. Assoc.*, Jan. 17th, 1903.

CLINICAL LECTURES

ON

DISEASES OF CHILDREN.

Lecture III.

THE DIGESTIVE DISORDERS OF INFANCY—COLIC AND VOMITING.

Delivered at the London Hospital, October 28th, 1902.

By ROBERT HUTCHISON, M.D.Ed., M.R.C.P.,

Assistant Physician to the Hospital, and to the Hospital for Sick Children, Great Ormond Street, W.C.

Gentlemen,—We come to-day to the consideration of the disorders of digestion in infancy, disorders which are due, in large measure, to neglect of those rules for the feeding of babies which I tried to bring before you in my last lecture.

I shall not distinguish between the digestive disorders of breast-fed and bottle-fed infants, although for obvious reasons such disorders are far commoner in the case of bottle feeding than in the case of breast feeding. Further, I shall not attempt to describe these disorders to you from the point of view of pathology or morbid anatomy, because to do so would be to assume a knowledge which we do not possess. I shall deal with them purely from the clinical or symptomatic standpoint.

The symptoms of digestive disorder in infancy are both local and general. The local symptoms are of three sorts—colic, diarrhœa, and vomiting—but I need hardly say that those different symptoms, although one describes them separately, very commonly coexist in the same case. The second or general group of symptoms may be comprised in the one term "wasting." To-day I shall deal only with the local symptoms. The general symptom—wasting—may be more conveniently deferred until we speak of marasmus as a special disease, digestive disorders being only one of many causes which may lead to it.

Let us begin, then, with that very common disorder, griping or colic. I think one can distinguish two causes as giving rise to the painful contraction of the intestines which we call colic. The first of these is acidity due to fermentation occurring in the milk, and producing lactic and other acids which irritate the bowel and induce over-contraction in its wall. The other, and

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perhaps the commoner cause, is the existence of undigested casein or curds in the intestine. I told you that the great difficulty in the feeding of infants with cow's milk is the indigestibility of the casein, and there is no commoner consequence of that than the production of colic.

How are you to recognise colic? Its chief symptom is pain. Of course, pain in the case of infants is expressed not in words but by cries, and whenever you find constant and frequent screaming it should always make you think of the possibility of colic, for that is one of the commonest causes of persistent screaming. There are, however, other causes for screaming which may lead you into error. One of these is *earache*. It is often difficult to be sure of the existence of earache in young infants; but in such a case the mother will probably tell you that the child has been in the habit of putting his hand to the ear or head, and this may indicate to you the seat of pain. Another possible cause of persistent screaming is *teething*, a cause which mothers certainly tend to exaggerate, but, nevertheless, it is one which you should always have in mind as a possibility. Teething and earache seem often to go together. Another cause, and one which is commoner than you think, just as teething is less common than you are apt to think, is *renal colic*. It is, perhaps, surprising to you to be told that little children suffer from renal colic at all, but the probability is that they suffer from it as frequently as grown-up persons do. Of course, I do not mean to say that children pass stones, but they do pass gravel. You may have seen in the post-mortem room the kidneys of quite young infants which contained uric acid infarcts, and even in fœtuses in utero you may find them. Another possible *cause* of screaming is *phimosis*, which gives rise to straining on passing water; and the pain which that entails, the result of what one may perhaps speak of as a colic of the bladder, expresses itself in screaming. Another group of causes which you have to think of when a child is constantly screaming is *tenderness of the bones*, resulting from scurvy, or possibly from rickets in its acuter forms, or from congenital specific disease. In that case the screaming will be more noticeable when the child is handled, or when it is put into its bath, and that, along with a careful examination of the bones, will enable you to establish or exclude this cause. There remains one other cause of screaming which you ought to think of when you have excluded every other possibility, and that is *mental deficiency*. One of the signs of defective cerebral development in young infants is screaming without apparent reason. You will therefore bear in mind those possible causes before you conclude too hastily that the child is screaming because of colic. But you must also remember that colic is perhaps commoner than all these other causes put together.

The *signs* by which you recognise colic are, in the first place, hardness of the abdomen. You put your hand on the child's abdomen and feel that it is unusually resistent; indeed, it may be almost knotty. You may feel little coils of intestine standing out, coils of firmly contracted bowel. And you will observe, in the second place, that as the child screams it tends to draw up its legs. The screaming may even be so extreme that the child passes into a condition of convulsion, for there can be no doubt that the irritation caused by undigested food in the intestines may be an exciting cause of convulsions in young children. Another sign by which you can be quite certain that the pain is due to colic is the cessation of the screaming on the passage of flatus. If that happens you may be sure that colic was the cause.

Those being the signs and symptoms by which you recognise colic, we now come to consider how you are to treat it. I shall describe first of all what ought to be the immediate treatment of an attack of colic, and then I shall ask you to consider with me by what means you may prevent the attacks coming back. The first thing to be done in an attack is to apply warmth to the abdomen. You can do that by friction with warm oil, by warm fomentations, or by poultices of linseed or mustard; and in very extreme cases it may even be necessary to use the mustard bath. In addition to the external application of warmth you can apply it internally with advantage by means of an enema of warm water. Inject two or three ounces of water, as hot as the child will bear it, well up into the large intestine. In addition it is well to give carminatives by the mouth to aid in the expulsion of flatus; peppermint water or dill water are suitable for the purpose. Some prefer to give small doses of sweet spirit of nitre, say ten drops.

By means such as these you will generally

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succeed in cutting short an attack of colic, and then you have to consider how you are to prevent its recurrence. One may divide cases for this purpose into two groups; first, those which are being fed by the breast, and secondly, those which are being fed by the bottle. If a breast-fed child is suffering from colic you will probably find on inquiry that it is being fed too often, or at irregular intervals. All that you require to do in such a case is to regulate the feeding—to feed every two and a half hours instead of every two hours, or every three hours instead of every two and a half, as the case may be. The tendency to over-feeding in those cases is a natural one, because the taking of warm milk into the stomach, just as the injection of warm water into the rectum, temporarily relieves the colic, and the mother, finding the child is relieved by suckling, tends to go on giving the breast too often, and so a vicious circle is set up; and what you have to set yourself to do is to break that vicious circle by seeing that the child is fed regularly by the clock, and not at irregular intervals. In some cases the colic of breast-fed children seems to be due to the casein of the mother's milk being either too abundant or for some reason more difficult than usual of digestion. When you meet with such a case you will sometimes find it of advantage to administer a few teaspoonfuls of lime water to the child before it is fed, the lime water being sweetened a little to make it acceptable to the child. That tends to prevent the child taking too much milk, and it dilutes the milk in the stomach, and I have occasionally found it serviceable. Further, you will remember to correct any constipation, which is so common in breast-fed children—a most important point. In breast-fed children colic usually goes with constipation, and in bottle-fed children with diarrhœa. How are you to treat the constipation? The wrong way is to give a violent and drastic aperient at irregular intervals, which is commonly done by mothers, who wait until there has been no motion for two or three days and then give a large dose of castor oil. Many of you, perhaps, do not realise how extreme constipation in breast-fed children may be. I have known such infants go a fortnight without having any action of the bowels unless something is given to produce that action. The proper way to treat these cases is, firstly, by massaging the abdomen. It does not matter much what you use to massage it with, so long as you prevent the skin being chafed or broken; a little sweet oil is as good as anything. The massage should be done round and round along the track of the colon in the morning and evening for five or ten minutes at a time. Some people recommend that you should massage with a preparation containing aloes. That is not necessary. It is analogous to the use of inunctions of cod-liver oil in wasting, a haphazard way of doing what you can do by another means with certainty—namely, by the mouth.

With regard to drugs, the proper aperients in these cases are those which have a tonic action on the bowel. What you want to do is to educate the child's intestine into acting spontaneously, and for that purpose you should select such a tonic aperient as aloes, with the addition of a little nux vomica. You will find some such formula as this useful:—tincture of aloes, 3—5 ♏ (the exact dose depending on the age of the child and the severity of the case); infusion or syrup of senna 20 ♏; tincture of belladonna 2 minims (to relieve any griping which the aloes may produce, remembering that infants stand belladonna very well); tincture of nux vomica ½ ♏ (infants stand strychnine rather badly) and compound infusion of gentian to a teaspoonful. Sometimes you can add with advantage a carminative water, such as peppermint water, dill water, cinnamon water, or anything of that kind which you please. Such a mixture should be given at regular intervals. In a mild case it should be given only night and morning; in more severe cases every eight hours, and in still more severe cases every six hours. Go on administering this mixture, irrespective of whether there is an action of the bowels or not, until the motions become more and more regular, and then gradually leave it off. Then you will find after you have left it off that the intestines can be trusted to act by themselves. There is one other aperient which I should like to mention, if for any reason the above prescription proves unsuitable—and the only such reason I know of is that children are apt to vomit it because it is not pleasant, and, containing as it does a bitter, it is very difficult to make it pleasant—and that is confection of sulphur. Half a teaspoonful of this may be given at night only, or both morning and night. It is an exceedingly efficient laxative for young children, and you will

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never have any difficulty in getting babies to take it; but it does not exert in the same measure as the prescription I have just written down, that tonic effect on the intestinal wall which it is your desire as far as possible to promote.

If colic persists in a breast-fed child after you have corrected constipation, the question will arise—are you to wean the child or not? Here I would remind you of what I said in the last lecture, that mere digestive disorder is by itself no necessary indication for weaning; it is only if the digestive disorder is accompanied by a persistent and steady loss of weight that you are justified in taking that step. So if, after correcting constipation, the weight still goes down, you may consider the question of weaning.

We will pass now to the commoner state of things which you have to deal with—namely, the prevention of colic in an infant who is being fed by the bottle. The great cause of colic in bottle-fed babies is a want of cleanliness in preparing the milk; and in particular it is the continuous use of those tube bottles of which I have already had occasion to point out to you the dangers. Such bottles produce far more colic, probably, than any other one cause. Your first duty is to see that the child has a proper and clean bottle; and, secondly, just as in the case of breast-fed children, see that the feeds are regulated, and not given too often. Your third care will be to see that the mixture which the child gets is sufficiently digestible. Assuming that the bottle is clean, and therefore, that fermentative action has not occurred, the colic may be put down to the indigestibility of casein. To correct that you have to give a mixture which contains less casein; in other words, you have to proceed from the positive to the comparative degree of digestibility, and put the child temporarily upon condensed milk. In very severe cases you may require to give peptonised milk. You may also require, as in the case of breast-fed children, to correct constipation. That you should do in the way I have already described. But more commonly you will have to deal with diarrhœa, and the best ways of treating that we shall consider when we speak of diarrhœa as a symptom of digestive disorder.

In all cases of colic, both in breast-fed and bottle-fed children, you will find the administration of a carminative between the feeds of help. A carminative mixture for a young infant should contain certain definite ingredients. First of all, it should be alkaline, and therefore you use bicarbonate of soda (2½ grains) as the basis. It should be alkaline because colic is so often due to acidity in the intestine. You supplement this with an aromatic mixture which is also alkaline; for instance, aromatic spirits of ammonia 2½ ♏. Secondly, to make this pleasant, you will add a little glycerine, 2 ♏. It is always advisable if you can to make medicines pleasant for infants, and one of the best ways of doing this is to add glycerine. Syrupy preparations are less suitable, because they are apt to ferment, and as you have already got fermentation going on you do not want to do anything to increase it. Thirdly, you make the mixture up to a teaspoonful with peppermint or some other carminative water. This should be given between the feeds, and it will be found useful in all cases. In addition, you should see that a child who suffers from colic is kept very warm. A common, but often unsuspected, cause of the disorder is chilling of the abdomen. Inquire, therefore, into the covering of the child's abdomen, and insist upon its having a broad flannel binder, even though it be beyond the age at which binders are used, and see that the binder does not slip up over the chest and leave the abdomen unprotected. See also that the feet and legs are kept warm, for cold feet are a very common cause of colic in young babies. The child should have warm socks, and, if necessary, a hot bottle to the feet. If, in spite of all you do, the colic persists, and if at the same time the infant is gaining weight (which sometimes does happen), so that you are not justified in making any great alteration in the feeding, you may perhaps require to have recourse to opium in some shape. You should not give opium any oftener than you can help, but sometimes you are driven to use it owing to the persistent pain which the child suffers. The best preparation to use is codein $\frac{1}{30}$ or $\frac{1}{40}$ grain, which, with glycerine and some carminative water, makes a very suitable opiate for a young child.

I now pass on to the consideration of *vomiting* as a sign of dyspepsia in young infants. There are two varieties of vomiting which you will learn to recognise when you get into practice as signs of digestive disorder. There is (1) *acute vomiting*, which is usually the result of an attack of

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gastric catarrh; and there is (2) *chronic vomiting*, which may be the result of chronic gastric catarrh, but is very commonly due merely to unsuitable feeding.

You will have no difficulty in recognising the acute type. The child will be brought to you with the history that it began to vomit a few hours before. You will find that the skin is hot, the tongue thickly furred, and that there is a sour smell about the breath. The only difficulty in your diagnosis will be the exclusion of more serious disease. Many of the acute specific fevers in children begin with vomiting; so do many intracranial diseases, and you may at first be doubtful whether you are not dealing with some such case, particularly as acute gastric catarrh is often accompanied by a high degree of pyrexia. You may, therefore, do wisely to reserve your diagnosis at first.

The proper treatment of acute vomiting is by *starvation*. You need never be afraid to starve young children in the matter of solid food. It is said that infants bear starvation badly. That may be true of chronic under-feeding, but children will stand the complete withdrawal of all nourishment for two or three, or even more days, without any disadvantage, and, indeed, often with great benefit, provided always that you fulfil two conditions. The first of these is that the child is kept warm; children stand starvation badly simply because they lose so much heat from their surfaces, a fact which is true of all young animals. And the second condition is that water must on no account be withheld. Children stand the withdrawal of fluid very badly indeed. So when I say that they are to be starved it means that they are to be given nothing which can be described as nourishment, but they must have abundant liquid; and in such cases as we speak of that may take the form of boiled water, or, if you like, and in order to satisfy the mother, one may administer a thin decoction of barley or weak veal broth. The liquid, whatever it may be, should be given cold with a teaspoon.

You will find also a great help in these cases from *washing out the stomach*. This is the first time I have had occasion to mention washing out of the stomach as a therapeutic measure in children. I can only say that you will find it of the very greatest use in all cases, acute or chronic, whether in breast-fed or bottle-fed infants, where vomiting is a prominent symptom. The method is so simple, it is so safe, and, compared with a similar proceeding in adults, so comparatively pleasant to the patient, that you need have no hesitation in employing it. You should use for the purpose a small size red rubber œsophageal tube. The œsophagus in a baby is bigger than you might think; it is certainly bigger than an ordinary pencil, and you will be able to get the smallest size of tube down it without difficulty. It is an advantage to use a tube which is rather large than otherwise, because the tendency of a small tube is to permit regurgitation of fluid alongside, which leads to coughing and choking, and to struggling on the part of the child. The œsophageal tube is connected by means of a piece of glass tubing to another piece of soft rubber tube, and that to a funnel such as I show you, which is really nothing more than the barrel part of a glass syringe from which the piston has been removed. A funnel of this shape has the advantage that you can blow down it if you want to. Sometimes the eyelet of the œsophageal tube gets blocked with a mass of casein, and it is then an advantage to be able to put your mouth over the top of the funnel and displace the casein by blowing. By the same means you can produce a sort of shower-bath in the interior of the stomach, and by projecting the fluid with some force through the tube you can wash away the mucus and masses of casein which have accumulated there. It does not matter much what fluid is used in the washing out. One usually employs plain water. You can use it quite hot, remembering that it always tends to get cooler as it passes down. But if there is a very sour odour in the breath, I recommend you to add a little bicarbonate of soda—a teaspoonful to the pint—before the final washing. Having completed the washing, you will find it an advantage to leave an ounce or two of fluid behind, for this helps to fulfil the condition which I have laid down, that the child must be kept supplied with a large quantity of liquid when it is being starved. The hot water also acts as a kind of internal poultice, and is soothing. After this manipulation a child will often go quietly off to sleep, though it may have been screaming for some hours before the washing out was done.

You will not find *drugs* of much assistance in acute vomiting. If you wish to have recourse to

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them, one would recommend especially small repeated doses of calomel, particularly if the tongue is much furred. One tenth to one sixth of a grain every hour for half a dozen doses is a suitable quantity for an infant; and that may be followed by the use of bismuth, either the carbonate or the subnitrate. Be sure always to give bismuth in large doses; that is to say, never less than ten grains, and even in a young infant you can safely give more than that.

The *chronic vomiting*, which is the result of digestive disorder, is of quite a different nature to that I have been describing; and I would make this first remark about chronic vomiting in young infants, that it is of no importance at all unless it be accompanied by progressive loss of weight. You are not to get anxious, as nurses and mothers do, because a child vomits after its feeds. Many children, like the Romans of old, have a way of swallowing more than they want and rejecting the excess, and that is not a habit which need alarm you. It is only when the vomiting is accompanied by progressive loss of weight that you should take it seriously. The second remark I would make is, that if you are dealing with quite a young infant,—that is to say, one of a few days, or at most a few weeks old,—and if the vomiting has dated from birth, you should make certain if you can that you are not dealing with a case of congenital pyloric obstruction. I shall have occasion later on to describe at some length the disease known as congenital hypertrophy of the pylorus in young children. It is a disease which is not common, but you ought to be able to recognise it; and it lends itself so well to surgical treatment that it is extremely important you should recognise it early. The main symptom of that disease is chronic and habitual vomiting, occurring practically from the moment of birth, and when you get such a history as that you should have its possibility before your mind.

Now, how should you treat that chronic form of vomiting which is the result of purely digestive disorder? The first thing is to regulate the feeding. Vomiting, if it is habitual, means that the child is getting a mixture which is too strong for it, and you must dilute the milk still further, or else give it in a more digestible form. And here, again, it will be extremely important to regulate the times of feeding, taking care that the bottle is not given too often. In these cases, also, you will find periodic washing out of the stomach to be of the very greatest service. Every day, or even oftener, you can employ it with the greatest benefit. With regard to drugs, I would only say again that they must take quite a subsidiary place. It is only after you have corrected the feeding and washed out the stomach thoroughly that you should think of drugs at all. You will find bismuth one of the most useful if given in large doses. In other cases, again, two or three minims of dilute hydrochloric acid, with a little glycerine and some carminative water, will be advantageously prescribed. I cannot tell you beforehand which cases will do best with bismuth and which with hydrochloric acid. I think it is more or less an experiment in every case; but one or other of these drugs will generally be very beneficial, supposing you have failed to relieve the child by adjusting the feeding and washing out the stomach.

Intestinal Indigestion and its relation to Arterial Sclerosis and Renal Disease.—Weber gives the clinical histories of three cases exhibiting the sequence of events named in the title. Commenting on the fact that children with cholera infantum frequently suffer and die from acute nephritis, he sees no reason why similar poisons continuously supplied should not produce chronic renal changes, especially those characterised by interstitial inflammation. The mode of action of these poisons is probably that brought forward by Ribert, who has maintained that the noxious agents coursing with the blood-current do not attack the parenchyma directly through the capillaries, but get into the lymph spaces, and, in flowing from the periphery toward the hilum of the organs, develop their deleterious influence upon both parenchyma and interstitial tissue. By the peculiar tract-like way in which the lymph channels and lymph nodules are arrayed beneath the capsula propria and within the cortical substance of the kidney, the focal distribution of the inflammatory process would then be explained. With reference to the treatment of these cases, the author says that it is self-evident that the administration of small doses of the iodides constitutes part of the treatment of established sclerotic disease; when disease of the kidneys is threatened through gastro-intestinal indigestion, careful diet, intestinal antisepsis, *i. e.* calomel in very small doses, frequent cleansing of the colon, regulation of the patient's life, etc., will be of direct benefit in our therapeutic measures.—*New York Med. Journ.*, Jan. 17th, 1903.

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INTERNATIONAL CLINICS. Vols. i and ii, twelfth series, 1902. J. B. LIPPINCOTT & Co.

The series of volumes published quarterly under the title of 'International Clinics' has now established for itself a firm position in medical literature. The clinical lectures form the largest and most important part of each volume, and are of varying quality, as must always happen when the contributors are numerous. Some have something to say, and others appear to work to order, or to have their remarks at an ordinary clinical demonstration taken down and published. Amongst the authors are several well-known men in America, Canada, England, France, and Germany, and the value of having the practices of these different countries described by able teachers, many of whom have made a special study of the subject on which they write, is undoubtedly great. Boas writes on habitual constipation; Hemmeter on gastro-intestinal auto-intoxication; Finger on acute urethritis; Jonnesco on resection of the cervical sympathetic; Robin on ulcer of the stomach; Senn on vesical calculus; and Pavloff on the function of the digestive glands. As text-book teaching must always lag a little behind actual current practice, one can learn in these volumes what are the latest views on pathology and treatment. A paper like that of Professor Jonnesco's, who during the last five years has performed over one hundred and thirty bilateral cervical sympathectomies, introduces the reader to one of the latest advances in surgery, described by its most famous exponent. Whether the reader will share the author's view that "resection of the cervical sympathetic will occupy a prominent place in the future progress of surgical science" is another question.

In addition to the clinical lectures and papers on clinical subjects, certain new features are apparent in the more recent volumes. One of these is a review of the progress of medicine during the year, and in this department there is a valuable article by Dr. Ravenel, who discusses Koch's pronouncement on the transmissibility of tuberculosis, and gives an account of the further evidence on this question which has recently been accumulated. With this exception the notes on the progress of medicine, surgery, and gynæcology are too short to be of value to any one, and it is impossible to give a comprehensive view of these subjects in the space allotted, about a hundred pages.

Another new feature is the biographies of living medical men, with such interesting details as their photographs, their hours of work, of sleep, and of recreation, their meal-times, and sometimes their meals, even their fees and the measures they take to secure them. It almost looks as if some sheets of the 'Strand Magazine' had got bound up by mistake with 'International Clinics.' Such excrescences are distinctly pathological in character, and the only sound treatment is complete excision.

Headache.—The various forms of headache are noticed by Leszynsky as the result of a clinical study of over 5000 cases during the last twenty years. In a large majority headache is due to toxæmia, either from constipation or intestinal or gastric indigestion. In these cases it is usually frontal, but may be general. Gastric indigestion may produce it reflexly through the pneumogastric nerve, and headache due to both gastric and intestinal causes is frequently an accompaniment of stomach dilatation. Headache due to renal disease may be either uræmic or congestive. The importance of urinary examination for uræmic headache is specially remarked on. There are two kinds of rheumatic headache, one due to sub-oxidation and autotoxæmia, and the other a muscular rheumatism or myositis of the scalp. The latter may be induced by exposure to cold, like other forms of muscular rheumatism. Gouty patients are specially subject to headache, as are also diabetics, and in them it is often accompanied by hebetude and depression of spirits. Headache is a common symptom of nearly all the infectious diseases and fever; is often due to the toxic action of alcohol, coffee, lead, etc.; and it is an important symptom of neurasthenia, though not always present. Its importance in epilepsy is at times very great, especially in diagnosis of obscure cases. He considers a constant general headache with optic neuritis and frequent attacks of vomiting, invariable signs of intra-cranial disease. [This is a rather general statement which should be considered open to exceptions.] Syphilitic headache is a matter of importance. He insists on the necessity of careful examination of the entire organism in every case to clear up the cause.—*Journ. A. M. A.*, Jan. 17th, 1903.

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A CLINICAL LECTURE

ON

EXOPHTHALMIC GOITRE.

Delivered at Guy's Hospital, November 1st, 1902,

By W. HALE WHITE, M.D., F.R.C.P.,

Physician to the Hospital.

GENTLEMEN,—We have in Miriam Ward, sent to the hospital by Dr. Donbayand, a woman suffering from exophthalmic goitre, and so I think we might this afternoon go over some points about the case. In order that you might learn from actual experience rather than from anything else, I went through some cases from my own case-books at home to see how the woman in the ward corresponded to other cases. The patient has kindly come to the lecture, and if you look at her you will see that she has the main classical symptoms—she has prominent eyes; she has very considerable enlargement of the thyroid gland, which you will notice is not quite symmetrical. And you must take my word for it that her pulse is very rapid. You will see she is a thin, wasted woman, and you notice there is a fine tremor present when she holds out her hand. Contrast that with the hand of a healthy person, and you will see that the tremor is very considerable indeed. And although there are many other points about the case, those are the only ones I can demonstrate to you here—namely, the prominence of the eyes, the enlargement of the thyroid, and the tremor and wasting, possibly with a certain amount of anæmia. You may say, cannot I demonstrate the symptoms connected with the lids? I cannot do that because she does not show them very well; but to me, as I am close to her, it is evident that the upper lid lags distinctly behind the eyeball when the eyeball descends. That lagging is known as von Graefe's sign. I do not think there is much retraction of the lids; that is Stellwag's sign. Possibly the

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lower lid is a little retracted. We will now send the woman away and then discuss her case.

The best symptom to take first is the enlargement of the thyroid. I will try to tell you a few practical points about each of the symptoms as we go through them. With regard to the thyroid, undoubtedly the most important thing to remember about it is that it may exist enlarged for a very long time—even as long as thirty years—without any symptoms of exophthalmic goitre supervening. You will notice the great importance of that, because it will make you hesitate to operate upon an enlarged thyroid if there is any possibility that the possessor of it may be a dormant case of exophthalmic goitre; for it is notorious that such patients bear operations very badly. Amongst my own cases which I have abstracted here is one that is a good instance of the long time which it lasted. It is that of a woman æt. 43, who says that she had her thyroid enlarged for twelve years before the supervening of any other symptoms of exophthalmic goitre; and in the cases mentioned by Dr. Pendred in a recent 'Guy's Hospital Gazette' you will see he draws attention to the same point. It is a point of immense practical importance, which you must bear in mind or else you will find yourself operating upon cases of dormant exophthalmic goitre, and very likely the patients will consequently die. The thyroid gland may be huge. The largest among my own series of cases was that in which the description says it was on one side as big as a fœtal head and on the other side as large as a Tangerine orange. Usually the right is the larger portion if there is any difference, but occasionally, as in one of the cases I have noted here, the left is the larger. It is important to remember that the size may fluctuate from time to time; and another thing which is very interesting is that a cystic adenoma may develop in the gland that is enlarged in exophthalmic goitre. One of my own series of cases shows that point. We know also, as the books tell you, that you can get a thrill over it; you can hear a systolic murmur, and you can hear a continuous venous hum. In some of the cases the thyroid may shrink. For instance, here are notes of the case of an elderly lady, a patient of Dr. Roe Carter's, and very many years ago—she could not remember how many—she was a patient of Sir Samuel Wilks for exophthalmic goitre; and at that time the thyroid was very big. When I saw her about two years ago the thyroid was even smaller than natural, but the eyes were still prominent. So you see the size may vary very considerably.

To take the eyes next, you will have noticed in our patient that the exophthalmos was bilaterally symmetrical. That is of very great importance. Occasionally one eye looks a little more prominent than its fellow, but I think you will be well advised if you hesitate a long time before you allow yourself to diagnose exophthalmic goitre if one eye is prominent and the other is not. Such cases are recorded, but they are very rare; and nearly always when one eye is prominent and the other is not, the patient is really suffering from a tumour at the back of the orbit, which is pressing that one eye forward. I remember not very long ago being asked to go into one of the surgical wards to see a case which some thought must be a case of exophthalmic goitre, a case of unilateral exophthalmos; but, bearing in mind what I have told you, I considered it to be a case of tumour at the back of the orbit, and so it was proved to be. It was operated upon, and the tumour removed. The exophthalmos may be very extreme. In one of my cases the insertions of the ocular muscles were visible. Then a very, very important thing is that exophthalmos may often be absent for a very long while. That follows from what I have said just now, that the thyroid may be enlarged without any other symptoms. You cannot over-estimate the importance of remembering that exophthalmos may be absent for years. For instance, here is a case in point. A lady æt. 35, a patient of Dr. H. M. Stewart's, undoubtedly had exophthalmic goitre, although she had no exophthalmos; there can be no doubt about the diagnosis, because she had the tremor, the enlargement of the thyroid, and the rapid pulse, her pulse being 150 to 160 per minute. The prominence of the eyes is liable to vary from time to time, and, as I said just now, von Graefe's sign—the failure of the lid to closely follow the movements of the eyeball downwards—is very often absent. So is what is called Stellwag's sign—that is to say, the retraction of the lids. The importance of Graefe's and Stellwag's signs has been much exaggerated, for both of them are often absent in cases of exophthalmic goitre, and both of them may rarely be present in other

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conditions. Still, they are confirmatory of exophthalmic goitre when they are present.

Passing to the next of the classical symptoms, the tachycardia, I do not think you can overexaggerate the importance of this, because it is the commonest symptom of exophthalmic goitre. It is probably never absent, and, generally speaking, it is the first symptom to be present. Here, for instance, is a case showing the importance of it as an early symptom:—A woman æt. 27 had influenza. She was regarded as a case of tachycardia following influenza. On going over her carefully a slight degree of exophthalmos was discovered—a slight enlargement of the thyroid gland and a slight tremor. I saw her again with her doctor a month later, and she was then a perfectly typical case of exophthalmic goitre. That illustrates well that tachycardia is the first symptom, that it is a constant symptom, and that it is of great importance in diagnosing the disease. You should invariably be on the look-out for exophthalmic goitre in any case of tachycardia in a woman. If you do that you will save yourself from many mistakes. Usually the pulse-rate is somewhere between 120 and 150. It may vary in the same case, and of course it is quicker when the patient is labouring under emotion or excitement. It is usually regular and usually small. The other circulatory symptoms are well described in the books. There is often a pulmonary hæmic murmur, often a systolic murmur at the apex which the general condition of the patient makes you think is hæmic, and the patient usually is anæmic. Usually there is a wide area of cardiac impulse, and there are signs of cardiac dilatation, such as the apex being at the nipple line or a little outside it.

The next symptom I want to draw your attention to is the tremor. I am sure, after examining candidates for many years, that the majority of students do not appreciate the importance of the tremor. It is the next commonest symptom to tachycardia, and it is one of the earliest to appear. It may be very marked, and it may, as in one case amongst these notes, be even present in the feet, though it is best seen in the hand. The reason I think students are not impressed with its importance is that they often fail to look for it. Perhaps the best way to demonstrate it to you is to show you this handwriting of a well-educated lady who could previously write properly, who wrote to me a letter in order that I might have an opportunity of preserving a record of the tremor. The whole letter shows very well the fine tremor right down to the signature. The tremor is often so great that the patient has what she describes as the fidgets. For instance, the following cases were marked instances of fidgets. In the case of that lady whose handwriting I have passed round the note is: "She is so fidgety and restless that her sister cannot sleep in the same bed with her." You will admit she must have been very fidgety. In another case the fidgets are again noted as being very prominently present. In another case also they are very marked. These three cases, seen consecutively, showed fidgets very well indeed. The tremor may be extremely severe. A good instance of that is in the case of Mrs. G—, an American woman. The note says the tremor is so great that she cannot unbutton her clothes. I remember it very well, because I had to unbutton them myself to listen to her heart. So these facts will give you some notion of the great extent of the tremor. I have some cases which show admirably its importance in diagnosis. For instance, one I mentioned just now with tachycardia. Her symptoms were tachycardia, some prominence of the thyroid, and tremor. The tremor clinched the diagnosis and showed the tachycardia was due to exophthalmic goitre. Another instance is that of the lady in whom the disease followed influenza. When I saw her first she had tachycardia; the prominence of the eyes was very slight and the enlargement of the thyroid very slight. It was the tremor by which I was able to convince her doctor that she was a case of exophthalmic goitre. Here are notes of a woman who complained of headaches for many years. She had seen many doctors and had been treated for various ailments. She had tachycardia and tremor, and she had had both for years. Recently the eyes had become a little prominent and the thyroid had become enlarged. If the tremor had been properly appreciated in that case the diagnosis would have been made years before it was. So much, then, for this regular fine tremor.

The next condition I want to draw your attention to is the mental condition of these people. I am sure we have to do with a mental condition in our present patient. These people are always more or less irritable; they are in a state of sup-

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pressed excitement. It has been described as the state which comes over candidates when they are waiting for the publication of examination results. These patients are very emotional, very restless; they cannot sit still in a chair five minutes together, they have to get up and walk about the room. That drifts in some cases to actual insanity. It is a well-known fact that the proportion of exophthalmic goitres in asylums is higher than it is among the community at large. To remind you of that, it is not long ago that we had to send a woman out of Mary Ward who had exophthalmic goitre because while in the hospital she developed suicidal tendencies. This mental instability, irritability, and condition of suppressed excitement are not nearly sufficiently impressed upon students in the accounts in the text-books. If you are on the look-out for them, it is very rarely that you will not see one or other of them in a case of exophthalmic goitre.

The temperature, I think, is always raised in a patient who is bad enough to come into the hospital. The rise of temperature is usually of slight degree—a little in the evening. It was a little raised in the patient whom you saw just now.

The next symptom I want to draw your attention to, again because it is not properly insisted upon, is the wasting. I think you hardly ever, if ever, see a case of exophthalmic goitre, at any rate a marked case, in a fat person. You noticed how thin that woman was. Not only do they waste, but they vary in weight rapidly. Listen to the account of this case—that of a lady æt. 23. She lost three stones weight in eighteen months, and two stones of this loss occurred in the first seven weeks. That is one of many cases to show the rapidity with which these patients waste.

The next symptom is anæmia. That is common; these cases are frequently anæmic. It used to be an aphorism of Sir Samuel Wilks—and a very good one too—that if you see a young girl of about twenty and you think she is suffering from chlorosis, and you give her iron and yet she gets no better, it renders the probability great that she is really suffering from exophthalmic goitre.

The next symptoms to which I want to draw your attention, because they are very interesting, are the skin symptoms of this disease. These patients are very liable to be pigmented, and the pigment usually, as is commonly the case in women, is most abundant round the eyes. Sometimes this pigmentation is so dark that the disease has been more than once mistaken for Addison's disease. I have seen that mistake made myself. That will impress you with the importance of looking out for pigmentation in these people. Sometimes the pigmentation takes the form of being an excess of freckling. Another point in connection with the skin is that these people frequently sweat; they sweat on the very slightest provocation, and they complain of it voluntarily. I have so many references here to that symptom that I do not think I need read to you any one of them in detail. In the first one it is noticed that the patient sweats a good deal, in another it is noted that she sweats excessively, and so on. One of the cases also showed a great liability to factitious urticaria, and some of them complain of loss of hair. But most women when they lose their hair complain of it, and whether more women lose their hair in exophthalmic goitre than other people I am not prepared to say. The sweating, however, I am certain about. It is a prominent and marked symptom which does not receive enough attention, and the same remark is true of pigmentation.

An interesting matter in connection with the sweating is the next symptom—namely, marked polyuria. These patients are very liable indeed to pass large quantities of low specific gravity urine, and consequently they have to get up in the night to pass it. Here is the case of a woman whose mother also had exophthalmic goitre. She sweated and had polyuria. Here is the case of a woman æt. 33 who complained very considerably of passing so much urine, and the specific gravity of it was very low indeed. Here is another case, in which Dr. Adeney tells me that the polyuria has persisted. I saw her two or three years ago, and she had exophthalmic goitre with polyuria. She got better for a time; but again she has got bad, and has her other symptoms of exophthalmic goitre. But through the whole trouble, whether better or worse, she complained of the polyuria. But there is a much more important form of polyuria than that in which the urine is of low specific gravity, and that is the polyuria which in some of these patients is accompanied by sugar in the urine. I have a very striking case of that noted here. I wrote to the doctor of the patient before the lecture to ascertain how she was getting on. Here is Dr. Ensor's answer:—"Thank you for inquiring about

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the patient with Graves's disease. She is pretty well again [it must be six years since I saw her], and has been so for the past eighteen months. The glycosuria persisted two years after you saw her, and then disappeared. The tachycardia remained for some time after the sugar disappeared. Then the heart gradually slowed down, but is not yet normal. The extreme irritability of temper has greatly improved, and her nervousness is much less. The exophthalmos and von Graefe's sign I think are still present, but withal she is practically well and can walk seven or eight miles without any fatigue. The thyroid is, I think, smaller." That is very interesting indeed, because it shows that even what you might think is such an important symptom as the glycosuria will actually pass away. There is no doubt that these people have genuine sugar in their urine, but whether they are diabetic properly speaking I am not prepared to say, because I have not had a case since our attention has been directed to the importance of oxybutyric acid as evidence that the patient is to be regarded as a diabetic or is suffering from glycosuria. The fact that these people will pass sugar in their urine in exophthalmic goitre has been observed for a long while. For instance, in the 'Lancet' of 1875 you will find an admirable case recorded by Sir Samuel Wilks of a woman who had urine of a specific gravity of 1036, with a lot of sugar in it; and it is interesting that she had another of the symptoms—namely, early hallucinations, showing the mental tendency. You will not be surprised to hear of the next symptom of exophthalmic goitre—namely, that the people who are afflicted with it may suffer much from thirst; as far as I have observed, it has always been associated with polyuria.

The next symptom is a curious one—namely, that the appetite may be ravenous. I remember that our present patient complained of a large appetite, and she also says she is very thirsty. The ravenous appetite is very interesting, because in the first place it occurs in far more cases than ever have glycosuria. In the next place, you will notice that in spite of the ravenous appetite these patients waste so. The ravenous appetite occurs even when they have not got that next symptom which I shall come to directly—namely, diarrhœa. So this ravenous appetite must be looked upon as an independent symptom of the disease, and merely another evidence of what you see in the wasting—namely, a profound alteration in the metabolism in exophthalmic goitre.

As I have just hinted, the next symptom I want to draw your attention to is the diarrhœa that these patients suffer from. This may be of very considerable degree. For instance, I jotted down what proportion Dr. Maude and other authors have put it at. One says 7 out of 38 cases; another says 12 out of 15; and another 8 out of 28; and still another 4 out of 9. Those figures will give you some notion. So many of the cases upon which I have founded this lecture have had diarrhœa that I shall not have time to read them to you individually. The point to remember about it is that it may be in the first place very severe. Many of these patients had their bowels opened ten times a day at least. The diarrhœa is apparently quite independent of what food they take; and what particularly distresses the patients is that it comes on quite suddenly. The woman may be quite well one day, and the next day she may have severe and marked diarrhœa, and may have to go to stool ten times a day. This of course pulls them down a great deal and makes them feel weak and ill, and confines them to their bed. But it generally passes off, and usually speaking the patients recover from it, even when it has been very bad. On thinking back of the cases I have seen, I can only call to mind one case in which the diarrhœa killed the patient. I heard of a case from a friend in which the diarrhœa apparently proved fatal, but the fact that I can only remember one such case in my own experience shows that although you might hardly expect they will get over it, they do; occasionally they are sick with it. Some authors have said that some people with exophthalmic goitre have a slightly enlarged spleen. That I have not been able to detect. These patients are certainly liable to œdema about the feet, and that is not surprising considering how ill and anæmic they are. A symptom which I have not come across, but which is not unknown in the disease, is that the patient may have ophthalmoplegia, both externa and interna. I will show you in a minute how important that is in view of the pathology. I shall also be able to tell you later on that they always get, as found at the post-mortem examinations, their thymus gland enlarged; and in some of the patients you can percuss that out,

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obtaining a little dulness in the first and second left intercostal spaces. That was not so in the woman you saw.

Lastly, these patients are bad sleepers, and often suffer from insomnia.

So much for the symptomatology of the disease. I have tried to bring out the symptoms in their relative importance. You probably want to know also something about the frequency of it. I find that of the cases seen outside the hospital 1 per cent. are cases of exophthalmic goitre; and I find that 4 per cent. of the cases of exophthalmic goitre are males. But that is in my cases; others give 6 per cent., 8 per cent., or 10 per cent. of males. So you will see it is a disease of women. It most frequently occurs between the ages of fifteen and thirty-five, but it may occur under fifteen. When I say occur, I mean dating the occurrence from the time at which the first symptom could be detected. The youngest I have come across are one of twelve, one of fourteen, and one of fifteen. Those statistical facts are not of much importance. But now I come to what I think is of very great importance, because it may be a key to the understanding of the disease. The first associated condition which I want to draw your attention to is that of rheumatic fever. What the meaning of the association is I do not know, but many authors have drawn attention to it. On going through my series I find that in one case it is stated a brother has it; in the next case a father has rheumatic fever; in the next there is much rheumatic fever in the family; in the next case the patient has had rheumatic fever; and another patient has also had rheumatic fever. So the interest of those facts is that not only do you find rheumatic fever in the patient herself, but there is a history of rheumatic fever in the close relations. One of the patients, as showing the rheumatic tendency, has had chorea. Another associated condition is that of mental disease. For instance, one note says of the patient that there is much mental disease in the father's family. The next one gives a history of much mental disease in the family; for instance, the father is epileptic, the mother and the patient are both hysterical, and there is an aunt who is insane. Another patient was in a most curious mental state, so that I should think by now she is in an asylum. And Dr. Savage has recorded cases bearing in the same direction. For instance, Dr. Savage says one case had an insane father; another had insanity and eccentricity on the maternal side; and still another had insane cousins. So it is like rheumatic fever in that the patients are not only liable to be insane themselves and have mental disturbances, but they also come of a mentally defective stock, and mental defect is common amongst their relatives.

Another curious and interesting condition associated with the disease is glycosuria. I have already brought forward evidence to show that patients themselves with exophthalmic goitre have glycosuria. But listen to this; here is one case where the father has glycosuria, and the present patient in Clinical Ward has one brother and one sister both with diabetes. So glycosuria, rheumatic fever, and mental disease are liable to occur, not only in the patients, but among their relatives.

Another point that is not sufficiently insisted upon is the hereditary point of view. Of the cases of which I have notes here, in the case of one the mother had the disease; in another the mother had the disease; and Dr. Pendred calls attention to the same fact in last week's 'Guy's Gazette;' and I remember a case in the hospital where two sisters had exophthalmic goitre. You will notice that Dr. Batty Shaw, of University College, speaking in a debate on the subject reported in to-day's medical papers, mentioned a case of a father and son both having it. Amongst my cases there is an example of two sisters having it. So you see it occurs distinctly in families, and is hereditary to a slight extent.

You will naturally, perhaps, next want to know whether I can tell you anything about the pathology of this remarkable disease. Since we decided to take this subject for a lecture a paper was read upon it at the Royal Medical and Chirurgical Society, and is reported in to-day's medical papers. And Dr. Murray, of Newcastle, mentioned that in at least fourteen out of a series of cases there had been a history of simple goitre present in the patient before the other symptoms of exophthalmic goitre developed. That bears out what I told you at first, namely, that frequently—and the books do not bring that out with anything like sufficient prominence—the sufferer from exophthalmic goitre has had simple goitre before the exophthalmic goitre appeared. Sometimes, too, you will find not only that, but that the parents or the brothers

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or the sisters of the patient have also had simple goitre. Dr. Maude, who has made a special study of exophthalmic goitre, and who knows more about it than anyone else, pointed out an interesting fact—namely, that although what I have told you is undoubtedly true, and although there is no part of the world where you can get simple goitre more than you do in India, yet exophthalmic goitre is virtually unknown in the natives there. I can only state the facts, and point out to you that the interpretation of them has not been arrived at yet.

The next etiological fact which is worth mentioning is influenza. I am certain that some cases can be dated from that. I have notes of at least three where the disease followed distinctly on influenza. For instance, here is one: influenza; exophthalmic goitre shortly afterwards. Here is another: a big thyroid and other symptoms since influenza. And then there is a third case, which I have already mentioned to you.

The next etiological factor is shock, and there is no doubt whatever about the causative value of this. For instance, among the cases which I have notes of here, Mrs. G— dates the whole of her trouble from the sudden death of her child; another lady dates the whole trouble partly from influenza, and also from the fact that her husband stole some money or forged a cheque, and absconded and left the country. Here is another one, Miss M. E—. The doctor writes to say that "there is no doubt that the whole trouble has followed her being jilted," and you will find Dr. Pendred in the 'Guy's Gazette,' too, has given other instances in the same direction. In the medical papers to-day you will see Dr. Rolleston mentions the case of a man who underwent great excitement or shock at the siege of Pretoria, and developed exophthalmic goitre. And in connection with that there is the interesting fact, which has been known for some time, that during the siege of Paris the number of cases of exophthalmic goitre among the inhabitants mounted rapidly. So, look at it as you will, there is no doubt that sudden shock has much to do with the development of the disease.

Coming next to the prognosis, some of the patients die quite suddenly. That is a classical fact which has been recognised for many years, but not so many as you might think die in that way. I personally can only call to mind two who have died suddenly. Still, it will occur. Some years ago I wrote to a number of patients who had had exophthalmic goitre and had left the hospital, to try and find out what had happened to them. The way it worked out was this,—that while it was impossible to say that of those who were dead any had died from their exophthalmic goitre, they appeared to be distinctly bad lives. One had died of mitral disease; one of an abscess; one of a gastric ulcer; one of phthisis; one of heart disease; and in two the cause of death was not known. Other people have had the same experience—namely, that amongst your hospital patients cases of exophthalmic goitre are bad lives; that is to say, their expectancy of life is not the same as that of people in general. But inasmuch as—as I shall have to tell you when we come to speak of treatment—the great thing is care and rest, and a freedom from worry and anxiety and excitement, amongst your private patients you will find that the outlook is very much better indeed. Among private patients extremely bad cases may recover. I have read you a letter from a doctor about the case of a patient who had glycosuria. And then here is a very bad case that recovered, a patient æt. 23, sent to me by Dr. Matcham. She first noticed her eyes were prominent five years ago. She had to give up work. She rested at Hastings, and was afterwards able to go back to work. Then she broke down again; but she rested again and got better again. And I might relate to you many cases to show that if the patients are able to keep from anxiety and worry and over-work they will do well. One of the most remarkable was a man brought to me by Dr. Howells, of Brecon. After I saw him he had severe cardiac failure and extreme dropsy, but after many vicissitudes he, by careful treatment, recovered. So the outlook is much better among private patients than it is among hospital patients. They may even get well, as the case I read you as an example of bad diarrhœa did, although her bowels were often opened ten times in the twenty-four hours. They may even get well after having had bad rheumatic fever and pneumonia, as was exemplified by a woman in Mary Ward four or five years ago. But you will ask me, do they get absolutely well? No, they do not; they never remain really fit and well; they are always a little below par; they are never strong and robust women; and that accounts for some

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authors saying they never get well. But they get well enough to go through the ordinary avocations of life, if they do not have to earn their living. Here is another patient I wrote about, I having seen her two years before. Dr. H. M. Stewart replies, "With regard to the patient, there is very little to report. I have only seen her about four times this year. She has not been much trouble. The last time I saw her was in August, when her condition was much the same. She can walk about two miles without fatigue; the pulse remains rapid, the thyroid has not got larger, and beyond some indigestion at times she jogs along with a quiet life." And that is generally the outlook of most of them. To show the same point I went through some cases, and I found one had it twelve years before she was seen; one had it six years; another had it six; one had it many years; and another had it five years. Probably all of them had really had it longer than that. So that, whichever way you look at the question, you see what happens is that the patients get up to a certain point at which they jog along. Occasionally they appear to quite recover, but that is the exception. Here is a letter from Dr. G. Carrell, who kindly answered my letter about another case. After saying what he had done he goes on to say, "She got quite well and remained so, and since has performed her duties without a break." But that, remember, is quite an exceptional case.

You will say, what will kill these patients? As far as I know, the only instances in which death is due to the disease itself are those in whom death is sudden, those in whom it is due to the diarrhœa that they may get, and those who suffer severely mentally. Dr. Savage regards the supervention of mental symptoms in exophthalmic goitre as of very bad prognostic significance.

Naturally, the next point is with regard to treatment. The following things have been recommended for the disease:—Cold externally, faradism, galvanism, belladonna, digitalis, iron, arsenic, bromides, iodide of potassium, convallaria, strychnine, chloride of calcium, salicylates, phosphates, thyroid gland, thymus gland, and supra-renal. For the purpose of this lecture I took two text-books and looked up the treatment, and found those supposed remedies that I have read out to you. As I found all those in less than half a minute, I thought it would be endless if I went any further, and you may take it as a rule that when an enormous number of remedies are recommended for any disease we do not know what the remedy really is. For my own part, I do not know any drugs that can be depended upon to do good. You find that doctors record cases and say that the patient did well on belladonna, or that the patient did well on digitalis, or on thymus, or on anything else which may be given. But we have no knowledge whether the patient would not have done as well if the drug had not been given at all. It is a striking fact that when I wrote to the various cases which left the hospital to ascertain what had become of them and asked what treatment they had adopted, the woman who had done best and had been able to go back and earn her living said that she had never taken any of the medicine the doctors had ordered for her. It is not that I do not want you to use medicines at all. Some, like mercury, quinine, opium, and digitalis, are admirable and invaluable when used for the conditions for which they are beneficial; but I think we should protest against believing that in exophthalmic goitre any medicine at present discovered is curative, or even strikingly beneficial. Do not, therefore, go away with the impression that you cannot do much for exophthalmic goitre. As a matter of fact, you can do a great deal for your patient, because you can put her in the way of living in such a manner as will make her much more comfortable and increase her expectancy of life. You can rest her, for bodily and mental rest is the great thing. You can prescribe that such patients are to be freed from all worry and anxiety. How can we hope to get the woman well whose husband has forged a cheque and left the country? While there is worry like that the patient will never do well. Not only have you to insist as far as possible upon bodily and mental rest and freedom from worry, but the patient should have plenty of fresh air. And another point which seems so important is that these people particularly require a change of scene. I have known this happen over and over again: you send a case of exophthalmic goitre away to rest somewhere, and she gets on well for a time—four months or so,—and after that time she seems to make no progress. Send the patient to another place, and she will go on well again. If the patient is very thin, of course you will feed her up. Such cases will never do well so long as they remain thin. If

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they are very weak and thin, put them to bed and feed them up, and very gently massage them. You will of course treat the other symptoms which arise, such as the diarrhœa and the vomiting. I think if you will go on those lines you will do better than you could with any special drugs whatever. To show the importance of rest, let us take one out of many cases. Here is the case of a patient who had the disease two years ago. She rested for two months and got better. She went back to her work again and got bad. She rested again, and again she improved. And I might quote many cases from these notes in the same direction. You know that very often we have discussed the operative treatment. The oldest operation was cutting the sympathetic, because it was believed that exophthalmos had to do with that. And so it probably has, but that operation has been given up, because it is only treating a symptom of the disease. But some few years ago it was suggested it would be good treatment to take away half the thyroid gland, on the supposition that the symptoms were due to intoxication from the thyroid gland itself. That was very frequently done, and it did great good in some cases. The reason why it should be abandoned is this:—Take, for instance, a recent paper by Starr, and you find that out of 190 cases 45 were not available for report, leaving 145. Of these 145 who had half their thyroid taken out for exophthalmic goitre, no fewer than 23 died immediately afterwards, or within a few hours of the operation. That is why it was given up, and my own experience coincides with this dangerous experience. The first of my own cases I will relate recovered admirably from the operation. She was a lady, a patient of Dr. A. E. Taylor's. Mr. Jacobson took away half the gland. The notes say that the patient was thirty-two, had influenza, thyroid enlarged, pulse very rapid; she had tremors and fidgets, and prominent eyes. Mr. Jacobson removed the right half of the gland and the isthmus, and the pulse dropped from 150 to 70. But I saw her six months afterwards, and I saw the same tremor, heard the same hæmic murmur at the apex, and the apex of the heart was in the same position as before operation. The exophthalmos, it was true, was less, the pulse was less rapid, and the patient said she was very much better and was glad that the operation had been done. But of the other two cases under my care which were operated upon about the same time—when the operation was first suggested—one died a few hours after the operation with the classical symptoms,—that is to say, the rapid rise of temperature and pulse, for the former became 107°, and her pulse reached up to 200; she became extremely excited, nervous, and restless, with great anxiety and distress, with profuse sweating, and finally died from collapse. She was in Mary Ward. Another case, which was in Bright Ward, died under the anæsthetic, and these patients take the anæsthetic badly. So my experience was three cases—one fair success against two deaths. Considering that, I naturally ceased to advise operation. You will agree with me that the subsequent figures published by Starr support me, and I think you will find that most people have abandoned the operation. To my mind it is unjustifiable to take away half the thyroid gland when, as a result out of 145 cases, 23 died immediately or within a few hours; because you must remember we have already seen that the disease itself rarely or never kills. Other operations can be done; for instance, there are cases in which a cyst in the thyroid gland has been drained and much benefit followed; and sometimes it is necessary to divide the isthmus. Here is a case in point. A patient, Miss W—, very bad in all respects; neck seventeen inches round, trachea pressed upon. Her father had glycosuria. She had tremor and diarrhœa, and all the symptoms. She was very ill indeed. Mr. Lane, who saw her with me, although it was at the time that the lobes of the thyroid were being removed for the disease, agreed with me that she was too ill for the operation, and, indeed, we could not persuade the anæsthetist to give an anæsthetic. So her isthmus was divided under cocaine. A year afterwards the notes say she is much better, but the thyroid and eyes and other symptoms are much the same. She has not got the dyspnœa which she had from the pressure of the isthmus upon the trachea. Fortunately I heard of her seven years after the operation, and her doctor wrote to say she was in fairly good health, and all her symptoms were much less. She had no particular drugs, and no lobe taken away; she only had the isthmus divided. She was a bad case, and yet she gradually improved and did fairly well.

I cannot, for want of time, say much about the morbid anatomy. All I can tell you about the

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thyroid is that it is in a condition of hypertrophy physiologically,—that is to say, the acini of the gland are branched and the cells are more columnar. The thymus is nearly always enlarged, and if you take your post-mortems carefully you will find Peyer's patches, too, are always enlarged. I do not know what the meaning of those facts is.

To sum up clinically, the great importance of what I want to bring before you is early diagnosis. Look out for the tremor and tachycardia, as helping you to that. In at least three out of my series of cases the diagnosis was missed. And here, for instance, is a good case published by Dr. Maude, who knows so much of the disease. In Case 3, seen by Dr. Wilks many years ago, his notes said then, "Palpitation, pulse 153, sweats, very hungry. I regard the case as exophthalmic goitre, without either goitre or exophthalmos." There the diagnosis was made years before the classical symptoms appeared from the fact that the patient was thin, that she had palpitation and pulse 150, and that she sweated, and was very hungry. Then he gives another case, which I have not time to read, in which there was no doubt about the disease, but again very little goitre and no exophthalmos were present. These ill-defined cases are often described in the literature of the disease as the *fruste* cases, from the French word which means "defaced," and is applied to coins. Remember that if you do not get on with a case of anæmia in a young woman, always suspect exophthalmic goitre.

I will now tell you our views about the pathology of it. We really do not know much about it. There are two views before us now as to what is the pathology of the disease. One is that it is essentially a disease of the thyroid gland, and that the patient is ill because she is intoxicated with either an over-dose of normal thyroid secretion or an abnormal thyroid secretion. The evidence for that view is this, that no doubt many people get better when half the thyroid gland is taken away. Feeding monkeys on large doses of thyroid gland gives proptosis, widening of the palpebral aperture, and some wasting. But you will notice it does not produce nearly all the symptoms of Graves's disease It has been said also, and truly, that just occasionally Graves's disease is followed by myxœdema, which is undoubtedly due to shrinkage of the thyroid. But it is very rare. Dr. Murray, in over a hundred cases, knew of only one instance. At the best I have seen only one possibly doubtful case. It is so rare that I am not sure that that evidence is worth much. I have notes of a case in which the thyroid was quite atrophied in exophthalmic goitre, but she did not develop myxœdema. The other reason for believing that the disease is due to hypertrophy of the thyroid is that there is a certain contrast between exophthalmic goitre, a disease with a big thyroid, and myxœdema, a disease with an atrophied thyroid. In the one, myxœdema, there is a low temperature, while in exophthalmic goitre the temperature is somewhat raised. The skin is dry in myxœdema, but exophthalmic goitre patients sweat freely and readily. Next, patients with exophthalmic goitre are wildly excited, while the myxœdematous patients are sluggish and apathetic. Next, the treatment of myxœdema is based upon the administration of the thyroid gland, whereas in exophthalmic goitre part of the gland has been cut away with benefit. To my mind the facts do not by any means prove that exophthalmic goitre is due to an over-secretion or perverted secretion of the thyroid body.

The other view is that the nervous system is in some way the cause of the disease, and the evidence for that is that in the first place it is commoner in the emotional sex and at the more emotional age. Of the first point we have brought before you many instances, such as the woman who attributed the onset of the disease to the death of her child, and the young woman who was jilted. Next I have pointed out its relationship with insanity. In the next place, some experimenters claim to have produced some of the symptoms by operations on the restiform bodies. Next, like any mental malady, it is improved by rest, especially mental rest. And a certain number of cases have been recorded in which there has been some post-mortem evidence that the bulb itself is diseased. And when you think of the exophthalmos and the tachycardia and polyuria and enlarged thyroid, you will see that it is theoretically possible that disease somewhere about the bulb might cause some at least of the trouble. I recorded one case, which I cannot stop to describe now, which suggested that the cause of the disease was to be found in the bulb. And others have been recorded, for example, by Cheadle, Raymond, and Müller; and

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only recently in the 'New York Medical Journal,' of June 24th this year, you will find two other cases recorded by Dana. In one of them there was softening of the pons, and in the other, changes in the nuclei of the bulbar nerves.

The last point is as to the possibility that the parathyroid gland may have something to do with the disease. You know that if the parathyroid glands are taken away the thyroid gland undergoes physiological alteration which renders it very like the thyroid gland in exophthalmic goitre. But the gland under these circumstances does not get large; if anything, it gets smaller. It is said that the excision of the parathyroids in animals will produce some retraction of the eyes, and that after a time, if the animal recovers, the eyes become prominent, and that there is widening of the palpebral fissures. At any rate, it is certain that until we have cleared up the question of the exact function of the parathyroid gland we shall not be in a position to give any certain explanation of the pathology of the disease which we have been considering.

CLINICAL LECTURES

ON

DISEASES OF CHILDREN.

LECTURE IV.

THE DIGESTIVE DISORDERS OF INFANCY (*continued*)—DIARRHŒA.

Delivered at the London Hospital, November 1st, 1902.

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GENTLEMEN,—In this lecture I wish to discuss with you the third of the symptoms of digestive disorder which I mentioned at our last meeting—namely, *diarrhœa*. I cannot exaggerate the importance of this disease as a scourge of infant life. Recent returns of the Registrar-General show that out of 22,000 deaths from diarrhœa, taking the country all over, 17,000 occurred in children under one year old. That shows you the frequency of this disease, and to what a tremendous extent it destroys life. And you will naturally ask, why are children so liable to it? I think there are three causes which render children particularly liable to suffer from diarrhœa. The first is that the sterilising power of the child's stomach is very small; it secretes but little hydrochloric acid, and organisms which are swallowed with the food stand a good chance of running the gauntlet of the stomach and setting up irritation in the intestine. The second is that children are fed mainly upon milk, which is anything but a germ-free fluid; and the third is that infants are particularly liable to chill, for the reasons I have already mentioned. These are the chief causes which render children subject to diarrhœa, and against these you must fight if you wish to prevent it.

Many of the severest forms of diarrhœa in young infants are probably due to specific micro-organisms. Take what is called *epidemic diarrhœa*, the familiar "D. and V." of the hospital receiving room, which is so common in the summer. That is almost certainly due to a specific microbe. Recent attempts have been made in America to isolate it, and it is believed that it will prove to be identical, or almost identical, with the bacillus which is the cause of epidemic dysentery. The growth of these micro-organisms is favoured by certain quite definite conditions. If you trace the admissions of acute diarrhœa in such a hospital as this in the summer, you will find that gradually as the weather gets hotter the number of such cases increases, but you will also observe the curious fact that for some time after the thermometer has fallen the admissions still continue numerous; in other words, the curve of epidemic diarrhœa follows approximately the ordinary curve of the thermometer, but not quite accurately. And if you investigate the matter still further you will find that what it does follow quite accurately is not the temperature of the air, but the temperature of the soil. It is when the thermometer immersed four feet in the soil begins to record a temperature of about 56° F. that epidemic diarrhœa becomes most common. In other words, this organism, whatever it is, grows best in the soil about that temperature. Another cause which greatly favours the development of diarrhœa is the existence of bad surroundings—exhalations of all sorts, foul drains, contaminated soil, overcrowding, dirty cesspools, dung-heaps. All these things contribute greatly to the production of diarrhœa; and the importance of these causes is so great that often in practice you

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will find it an advantage to remove a child, if it is suffering from diarrhœa, right away from its surroundings altogether, and in particular to keep it out of large towns. I remember, when in Boston, going to see the Babies' Hospital there, which is kept specially for cases of summer diarrhœa. It is situated on an island down at the mouth of the harbour, and it is found that cases taken out of the slums of the city and removed to a place like that, where the air and soil are pure, often recover at once, without any additional aid. Further, you will be able to do a great deal in the prevention of diarrhœa by seeing that the milk is boiled before the child gets it, at least in hot weather. You will remember I told you that the importance of boiling milk is to some extent exaggerated. I wish now to qualify that statement by saying that the importance of it cannot be exaggerated in hot weather. All milk given to children in the summer should be boiled.

The third way in which you will be able to do something to prevent diarrhœa is by guarding against chill—seeing that the child is properly clothed, and setting your face against that foolish practice termed "hardening," which some mothers will try to get you to favour in the case of their offspring.

By such means as these you will be able to do a great deal in the prevention of diarrhœa. But in spite of all you do there is no doubt you will have a great many such cases in your practice, and therefore we may now pass on to a closer study of this disease and of the treatment appropriate to it.

In classifying the varieties of diarrhœa met with in infants I shall proceed upon clinical rather than upon strictly pathological lines, and I shall divide the cases—first, into those which are acute; and secondly, into those which are chronic. I shall distinguish among the acute diarrhœas three varieties—those which are simple, those which are febrile, and those which are choleraic,—and I shall make no subdivision of the chronic diarrhœas at all. When we come to treatment we shall take both acute and chronic cases together.

If the diarrhœa be acute it may be simple,—that is to say, it may be diarrhœa and nothing else; or it may be accompanied by pyrexia, when we speak of it as febrile; or it may be so severe in its symptoms, and accompanied by such profuse watery evacuations, that it is described as choleraic. The most dangerous form, what is called epidemic diarrhœa, the familiar "D. and V." of the hospital receiving room, is usually febrile in type. Sometimes in very severe cases it may be choleraic. Of course this classification is not by any means hard and fast. Diarrhœa cannot be definitely marked off into different classes; the varieties pass into one another. A case which begins as a simple diarrhœa may become febrile, and from that may become choleraic; just as it may be choleraic from the first, or may begin with fever. Or, on the other hand, it may remain simple throughout. You cannot always refer a case exactly to one or other of these classes.

What are the symptoms of infantile diarrhœa? In the first place, it may begin either gradually or with great suddenness. It may come like a bolt from the blue, affecting a child which was, or appeared to be, in perfect health. The motions are noticed to be more frequent, and often there is more or less vomiting. At first the stools are natural in colour, that is to say more or less yellow, but before the diarrhœa has lasted long they become greenish, and finally they may come to contain mucus, or even blood. If the diarrhœa still persists the motions become extremely offensive and of a watery consistence. I wish, at this point, to say a word or two about the character of the motions in different cases of diarrhœa, because I know it is a matter which will be apt to give you some trouble when you get into practice. What is the significance of the different kinds of stools which you meet with in the diarrhœas of infancy? First about the green stools, which are so common, and which resemble in appearance chopped parsley or spinach. What is the cause of their greenness? It has been supposed by some that the colour is due to the growth of a special micro-organism which produces a peculiar pigment. I do not wish to deny that that may sometimes be the case; but in the majority of instances I think it is merely due to the bile pigment being hurried on through the intestines without having undergone the usual changes. What is the meaning of the motions being stinking and putrid? That means that peristalsis in the upper part of the tract is too rapid. The consequence of this is that the food is hurried on before it can be properly digested and absorbed. You get, therefore, a mass of imperfectly digested and incompletely absorbed food

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lodged in the colon, and putrefactive organisms grow in it; hence the putrid stools. There is another variety of stool, which may be described as lumpy or cheesy, where you see white particles, varying in size, more or less oval or rounded in shape, scattered all through the fæcal matter. What is the significance of that? These round or oval particles are the remains of undigested milk; they consist partly of casein and partly of a soapy substance formed from the fat of milk and lime salts; and they indicate that the child is getting more milk than it can digest. Then there is a kind of stool in children which is extremely irritating, and which scalds the skin. If a child has that form of diarrhœa it gets an erythematous eruption over the buttocks, and I have seen those stools so irritating that a drop of them falling on the dorsum of the foot produced a blister in a few minutes. To what do these stools owe their irritating properties? It is due, I think, to the development of fatty acids—butyric acid and its allies. Then there is a stool which you may describe as "slimy;" that means a stool which contains mucus in excessive quantity. If you recognise excess of mucus you are justified in believing that the large intestine is particularly involved. You will remember from your histology that the large intestine is lined mainly by mucin-forming cells, whereas in the small intestine such cells are rather scanty. Therefore you will be justified in thinking that it is the large bowel which is at fault if there is much mucus; and if blood is passed in the motion, as it sometimes is, that suspicion amounts to a certainty. Such is the significance of the chief varieties of stools which you will meet with in the diarrhœas of infants.

We will now pass on to look at some of the other symptoms. The general symptoms may be trivial, as they usually are in simple diarrhœa; or they may be extremely severe, as they always are in the choleraic form. Where the constitutional symptoms of diarrhœa are severe you are justified in concluding that you are dealing with bacterial poisoning. There can be no doubt that the extreme collapse and depression from which those children suffer is not the consequence merely of the draining away of fluid from the body, although that helps in their production, but they are a direct result of poisoning by the absorption of toxins from the alimentary canal. Those symptoms may be described as the symptoms of collapse; the fontanelle becomes depressed, the eyes sunken and staring, and by-and-by they are kept partially open while the child is asleep, and you can perceive mucus on the cornea. The skin becomes wrinkled, the child seeming as it were to shrink away from its skin, which becomes redundant, and when you pinch it up, instead of falling back again as it would normally, it is apt to remain in folds. When you get this condition, which is described as a want of elasticity in the skin, you can be certain you are always dealing with a severe case. It is one of the points always to be looked for in your cases of infantile diarrhœa. Many observers believe that when such a condition of the skin is present you are dealing with a case in which there is an involvement of the kidneys, and in which partial suppression of urine is taking place. And unquestionably, if you examine the urine in those infants, you will usually find that it contains some albumen. The importance of recognising this want of elasticity in the skin is based upon the fact that when you find it you should institute special means of treatment, which I shall describe to you later. If the diarrhœa goes on, the symptoms of collapse become more pronounced. The temperature, which was at first perhaps high, falls, at least on the surface of the body, though it may remain high in the rectum; the child becomes blue and livid, and finally dies, death being often preceded by convulsions. That is the usual picture of a typical case of diarrhœa of severe type in a young child. I need hardly say, however that the picture varies a great deal. In the simple form there will be little evidence of collapse at all; in the choleraic form the signs of collapse will dominate the whole scene; whereas in the febrile cases the depression may not at first be very great, but becomes more marked at the close.

We may now pass to the *treatment* of acute diarrhœa. Here, gentlemen, I would wish to give you this piece of advice: Put not your faith in drugs. Your watchwords in the treatment of acute diarrhœa in an infant must be two; one is *starvation*, the other is *elimination*. If you bear in mind these two great indications, you will never go far wrong in the treatment of your case.

A word about each of those indications. You must starve the child because you do not wish to furnish any further pabulum for the growth of

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micro-organisms; and milk in particular you must withhold, for there is reason to believe that milk in such cases is actually poisonous. In a case of acute diarrhœa, whatever you do you must stop milk. With regard to elimination, your idea should be, in the first place, to remove as far as you can those organisms which are still growing in the alimentary canal, and in the second place to get rid of their poisons.

How are you to carry out those indications in practice? *Starvation* is easily carried out by withholding food; but, as I have told you before, infants only bear starvation well if you keep them warm, and at the same time supply them with plenty of liquid. Withhold all food for twenty-four hours in such a case, and give sips of boiled water, or a weak decoction of barley water. Or, if you want to make a pretence of giving some nourishment, give a weak veal broth or egg-white dissolved in water. Even in that case, however, I think the benefit is derived from the water; I think it is a matter of indifference whether you put anything in it or not. Still, as I say, you may wish to defer to the wishes of the parent in the matter of appearing to give something nourishing. If the case is not doing well you can continue starvation for two, or even three days. As the diarrhœa abates you may begin to give a little nourishment in the shape of whey, but the thing which you should give last, and be most careful about giving, is the casein of milk, because that is what does harm more than anything else. You may add raw egg-white to the whey, and continue this mixture until the motions are satisfactory.

With regard to *elimination*, if there is vomiting, begin by washing out the stomach in the manner I described in my last lecture. If the diarrhœa be severe, and especially if there is reason to suppose that the large intestine is chiefly involved, wash out the colon also; that is to say, begin your treatment by washing out "at both ends." I have already described to you the technique of washing out the stomach, and I would like now to say a word about washing out the bowel. I do not think it matters much what solution you use for washing out; warm saline is as good as anything else. It should be given through a douche-can or funnel. Care must be taken not to hold the can too high; it should never be raised more than two feet. Remember it is not difficult to rupture the colon of a child in attempts to wash out the bowel. I think it is best to have the child lying on its back, with the hips slightly raised, so that the fluid can gravitate down into the colon, using a small-bore soft œsophageal tube, just as for washing out the stomach. The tube should be introduced carefully, and you will find its introduction much more easy and satisfactory if you keep the fluid flowing all the time. The reason for this is, as you will readily see, that the colon being only a potential cavity and its walls more or less in apposition, the liquid as it runs in clears a way for the tube, and so you are less likely to get the end of it entangled in folds of mucous membrane, or to push it through the wall of the bowel. Pass the tube up as high as you can—you cannot pass it too high. Irrigate the intestine thoroughly, until the washings come away clear. By so doing you will wash away some at least of the organisms which are growing there. You also wash away a certain amount of the toxic products which these bacteria have been producing.

In order to favour elimination you should have recourse in all severe diarrhœas to subcutaneous injections of fluid. In cases accompanied by collapse, especially in those in which the skin is inelastic, elimination is apt to be interfered with simply from the fall of blood-pressure, and in such patients you have to make up the volume of fluid in the circulation as quickly as possible. The best way of doing this is to give a subcutaneous injection of sterilised normal salt solution, as many ounces as you can get in under the skin of the flank. It is very quickly absorbed, raises the blood-pressure, and helps to wash out poisons through the kidneys. Now you cannot, of course, wash out the small intestine, where the micro-organisms are chiefly growing. In order to promote elimination there you require to have recourse to drugs. In other words, paradoxical though it may seem, you must treat diarrhœa, in its early stages at least, by purgatives, just as you treat dysentery in grown-up persons by aperient drugs such as sulphates. You may start with a full dose of castor oil. If there is vomiting, and there is a difficulty in getting the oil retained, give a grain or two of calomel. When that has acted you have done all you can to clear away the putrefactive material from the small bowel.

There are still some special indications to be

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met. If the skin is in the inelastic state I have described, you will find it advantageous to give the child a wet pack, and you will find benefit also from the administration of diuretics, especially sweet spirit of nitre. If the signs of collapse are great, you may require to have recourse to the mustard bath, which is perhaps the most powerful stimulant which we possess. I like also to administer camphor in preference to alcohol in such cases, either in the form of a solution in olive oil (1 in 15 or 1 in 30), of which five minims is injected subcutaneously, or in the form of spirit of camphor, 5 to 10 drops by the mouth.

During all this time the child must be kept under the best hygienic conditions possible. It should be placed in a well-ventilated room, and should be kept very clean, the napkins being changed as soon as they are soiled, and never allowed to stay in the room after being taken off the child. In bad cases and in very hot weather the child should, if possible, be removed from the town to the country.

I said you were not to put your faith in drugs in the treatment of infantile diarrhœa. But of course drugs can give you some help, and I now want to say a few words as to the use and scope of drugs in the treatment of such cases.

Although, as we have seen, intestinal putrefaction is an important factor in the production of diarrhœa, I do not think you will get much benefit from the so-called intestinal antiseptics. I am no believer myself in salol and all the substances of that class, which are supposed to prevent intestinal putrefaction. I believe the best intestinal disinfectant is a dose of calomel, though whether it acts by virtue of its being calomel or because it is an aperient I do not know.

You may derive certain indications for the use of particular drugs from the character of the stools. If the motions are very sour it is well to administer alkaline remedies, carbonate of bismuth or aromatic chalk being amongst the best. If, on the other hand, the stools are very slimy and alkaline, many believe in the administration of hydrochloric acid. In the severe cases, so long as the stools are green I recommend you to give small doses of castor oil in some such form as this:

Ol. Ric.	℥v.
Mucilag. Tragacanth. . .	q. s.
Aq. Menth. Pip. ad . .	ʒj.

Give this at intervals of two or three hours for one or two days. Instead of castor oil you may give calomel and Dover's powder, $\frac{1}{8}$ grain of each. Later on, when the stools have begun to improve, you should give bismuth, remembering the rule to give it in large doses. A very favourite prescription, and one which is now in the hospital pharmacopœia, is carbonate of bismuth gr. x, calomel gr. $\frac{1}{6}$, Pulv. Ipec. Co. gr. $\frac{1}{6}$. Such a powder given every four or six hours, as the case may require according to its severity, will often be of great service to you after the acuter stage of the illness has passed off. If the stools continue to be watery and offensive, you may require to have recourse to astringents of some sort. You will find in nitrate of silver a very efficient aid. I regard it as my last card in the treatment of diarrhœa, and keep it up my sleeve, as it were, until the end; and there are not many diarrhœas which will withstand it. One sixth to one eighth grain of nitrate of silver is usually sufficient for a young infant. It may be given with dilute nitric acid, one minim; glycerine, five minims; and distilled water to two drachms. Such a dose, given three or four times in the day when the motions are very watery and offensive in the later phases of the illness, is often very useful.

As opium plays such a large part in the treatment of diarrhœa, I want to lay down to you, as clearly as I can, some rules for its administration. You can do great harm with opium; you can kill children with it quite easily. The warnings of the text-books in this matter are quite right. I have known a baby rendered dangerously comatose by one drop of laudanum. On the other hand, there is no one drug which is capable of rendering you such good service if properly used, and in many cases of infantile diarrhœa it is indispensable. Let me state, therefore, the following *rules for the administration of opium*, which I think you will find useful in practice. First, never give it at the outset of the illness. Begin with aperients, not with anything which will cause the contents of the bowel to be retained, as opium does. Secondly, never give it if there are signs of collapse, because then its narcotising effect becomes extremely dangerous. If the child is collapsed and cold therefore, be very careful in its use, or avoid it altogether. Thirdly, never give it if the tongue is furred. These rules, like all rules in medicine, are

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subject to certain exceptions; but when you are beginning practice let me advise you, if you want to avoid making serious mistakes, to keep them in mind. You will now require some rules as to when you *are* to give opium. Always give it if the stools are very frequent and accompanied by much straining,—that is to say, cases in which the lower part of the large bowel is particularly involved are those in which opium is specially indicated. Secondly, if the stools are offensive with a clean tongue, you have a combination of things which cries out for opium, because, as we have seen, this is due to the hurrying on of the contents of the small intestine by too rapid peristalsis. In such a case opium calms down the peristalsis and so prevents putrefaction and diarrhœa. Thirdly, you should always give opium in those cases in which the diarrhœa is of the lienteric type,—that is to say, in which a motion tends to occur immediately after food is introduced into the stomach. Diarrhœa of this type is due to an exaggerated excitability of the whole intestinal tract, and opium is the only agent which can be depended upon to lessen this excitability.

Two practical rules remain. One of these is, never wake a child up to give it a dose of opium. If you observe that rule you are not likely to produce poisoning. The child will sleep off an overdose of opium if you will let it; the danger is that the mother or nurse may wake it up to give it another dose when it is already in a drowsy condition. The remaining rule is that, as far as possible, you should avoid adding opium to other medicines, but give it by itself; that is to say, measure out a drop or half a drop of the tincture —for the liquid preparations are the most convenient—and add it to the other medicine when it is administered. The reason for this is that one often wants to withhold opium while going on with the other remedies.

The treatment of *chronic diarrhœa* I need hardly describe, because broadly it is conducted on the same general principles as that of an acute case. You do not require to starve such patients, but you select a suitable diet, prevent chill, and choose one of the drugs I have mentioned, according to the indications furnished by the stools.

There remains a special form of diarrhœa which I have not yet mentioned—namely, *ileo-colitis*. I speak of it separately because it is different from the other forms I have described in so far as it tends to be accompanied by ulceration, and to be confined to the large bowel and the lower end of the ileum. It may start as ileo-colitis, or any diarrhœa such as I have been describing may end by passing into a condition of colitis and ulceration. One reason why this disease is of importance is that it is apt to be mistaken for another important disease—namely, intussusception. You will be apt to diagnose colitis as intussusception, and *vice versâ*, and that is a mistake which is fraught with serious consequences. For this reason I bring forward the consideration of ileo-colitis separately. How shall you recognise it? Its chief features are that it is accompanied by fever, by straining or tenesmus, and by the passage of blood and mucus. How shall you tell it from intussusception? In intussusception there is less tendency to fever, there is usually vomiting, and you can generally feel the tumour either through the abdominal wall or *per rectum*. Mr. Barnard has pointed out a diagnostic sign of some value in distinguishing these two conditions. It depends on the fact that the contents of the small intestine are unable to pass the seat of obstruction in intussusception, so that if you get any bile on the napkins, or on the finger after it has been passed into the rectum, you may conclude that you are dealing with a case of colitis and not of intussusception. If your minds are open to the possibility of making this mistake you are not likely to fall into it often, but I have more than once known the abdomen opened in colitis under the impression that it was a case of intussusception, and I have known one case of intussusception which was allowed to die under the idea that it was one of colitis. So there is a real danger of the mistake being made.

About the treatment of colitis there is little to say; I have practically described it in speaking of the treatment of diarrhœa in general. As the motions are frequent and accompanied by tenesmus, opium should be given almost invariably, combined with castor oil at first, and later with large doses of bismuth. In this form of diarrhœa you will find washing out of the bowel particularly useful, seeing that the colon is the main seat of the affection. Wash out with warm saline. Some recommend a solution of nitrate of silver, but I think it is of doubtful advantage, and is apt to produce pain.

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WITH MR. STANLEY BOYD IN THE SURGICAL WARDS OF CHARING CROSS HOSPITAL.

May 15th, 1902.

Resection of the Shaft of the Radius; Replacement of it by Decalcified Turkey Bone.

THIS first case is interesting from several points of view. The boy is seven years of age, and, according to the imperfect history we obtained, broke his right forearm two years ago, and was taken to a London hospital, where it was set. Splints were kept on for five weeks. After a week without splints the forearm began to swell; apparently this swelling continued and, I presume, increased for a considerable time. This brings us to an uncertain date, when the boy was taken into the hospital he was attending with the diagnosis of myeloid tumour of the shaft of his right radius. He was operated upon by erasion; the posterior surface of the radius was cut down upon, the shaft opened, and the growth was enucleated from the centre of the shaft by gouges and sharp spoons. Microscopical examination of what was removed, as we were informed by the registrar, substantiated the diagnosis of myeloid sarcoma. After that he was in attendance at the hospital for some little time, and then the diagnosis was made that there was a recurrence of this growth. The boy's father was told in common language that the boy had a "cancer" of his armbone, and that an amputation would be necessary at the elbow. The father did not like this prescription, and brought him here. We found that the patient had a fairly regular ovoid swelling of the centre of the shaft of the right radius; over it, on the dorsal aspect of the radius, there was a scar about two inches long, which was slightly adherent to this deep swelling. The diagnosis of recurrence, of course, depended upon the observation of those who had watched the case

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all through. I accepted it, perhaps, too easily. It seemed to me, as the growth was known to be a myeloid sarcoma, which is of very low malignancy, and which is often treated successfully by erasion, that there was no particular hurry for amputating this boy's forearm; that in this situation it would be easy enough to deal fairly radically with it by excision of the shaft of the bone; and that if this treatment failed, amputation could still be performed —there being no danger of generalisation of this growth. So I suggested to the boy's father that I should excise the shaft of the radius, and endeavour to replace it by something which would give the boy a useful forearm.

You will have noted that this case was unusual in that it was the centre of the shaft which was involved, both the epiphysial ends being free from disease. Myeloid tumours affect cancellous bone as a rule, and in the radius the lower epiphysis is its usual seat.

Thinking that the recurrence might involve the scar of the first operation, I excised this, and then lengthened the incision upwards and downwards between the supinator longus and radial extensors of the wrist on the outer side, and the extensor of the fingers on the inner side, until it was only a little shorter than the radius itself. Then I raised the periosteum from the bone, completely separating it, first, from the narrow upper end, just below the tubercle. Here I divided the bone with forceps. It was now easy to shell the bone downwards out of its periosteal sheath to about three quarters of an inch above the epiphysial disc, where I again cut through the bone *in situ* with an osteotome. The whole excision being subperiosteal, no tendons were interfered with or even seen. I had next to replace the bone by something which would act as a spanner, and hold the epiphyses apart from one another whilst the periosteum laid down new bone. Naturally I chose for that purpose decalcified turkey-bone drainage-tubes. Fixing two of them together, I was able to get the exact length of the piece of bone I had excised. I put it into the sheath of the periosteum, hitching it against the lower epiphysis below, and above inserting into its lumen the stump of the shaft of the radius where it had been divided below the tubercle. Several stitches brought the periosteum loosely around it, fixing it quite securely in place; the deep fascia and skin were closed over all. A splint was applied, leaving the fingers free. The boy has never complained of anything. You see the wound has healed, and he has perfect use of his wrist, fingers, and thumb. He can shut his fist, open his hand, shake hands with the splint off, supinate from full pronation to beyond the mid-position, though it is only a fortnight since the operation was done. I do not know what will happen to the shaft; but it seems to me that there is a very fair probability, in a young growing boy, that there will be new bone thrown out by the periosteum, and that, as the turkey bone is absorbed, a fresh bony shaft will be formed. If we do get this it is difficult to see why the radius should not go on growing, for I have not interfered at all with the epiphyses, nor with the bone near the epiphysial discs. I have divided the artery of the shaft of the radius; the supply for the epiphyses is separate from that for the shaft; so all that has gone is some of the blood-supply from the shaft side to the epiphysial disc. It will be very interesting to see what will be the late result of the case. I trust it will be entirely satisfactory; for I was much distressed to find on section of the removed shaft that its swollen portion contained a cavity filled with fibrous tissue, in which were a few spaces containing remains of clot.

Large Cyst occupying the Cavity of the Greater Omentum.

Our next case is a boy æt. 11. There is no family history of tubercle, or of anything bearing upon the case. With regard to his personal history, he has had no illness except measles, and was always in good health until the commencement of this trouble, which began in January of this year. The statement is that he then began to feel very sick, and that he had occasional attacks of vomiting. He lost his appetite, and was unable to run about because he felt so tired. The abdomen began to swell at the same time, and it has increased steadily in size from then to the date of admission. He was admitted under my colleague, Dr. Abercrombie, on March 3rd. For the past month he has been having pain in the lower part of his abdomen, but it has never been severe. He has lost flesh considerably. You see he is now very thin and somewhat pale, though six months ago, apparently, he was an ordinarily nourished child. Before operation his abdomen

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was greatly and uniformly distended, so that it hampered the chest movements, and pushed the heart apex up to the third space just outside the nipple line. There was a soft resistance to pressure all over the abdomen, so that you could not press a hand back into the cavity of the abdomen; but no outline of any tumour or cyst could be felt. There was a most obvious thrill or wave in all directions; indeed, a more obvious or superficial thrill could not be conceived. No masses of any kind could be felt; the consistency of the swelling was absolutely uniform. It was dull everywhere and in all positions except in the right loin. The only change noted was a steady diminution of the resonant area. When the boy was admitted under Dr. Abercrombie, diagrams made at the time show that in dorsal decubitus not only was the side of the right loin resonant, but even a little bit of the front was so. When I saw him about the beginning of May—he had been admitted in March—resonance in dorsal decubitus had disappeared; the whole abdomen from pubes to xiphoid and from loin to loin was absolutely dull. If one turned him over a little bit to the left there was always resonance above and below the right twelfth rib, though dulness persisted anterior to this. When turned quite over on to his face an area smaller than the palm and occupying the region just mentioned was always found to yield a low-pitched tympanitic note, such as the stomach might give. Below this a short, very high-pitched note was obtained along the course of the ascending colon. The appetite was fair, sickness had ceased, the bowels acted regularly. There was no swelling of the feet. The temperature was somewhat variable. Occasionally it reached 100°, or even a little higher. At first sight the chart seemed to show a hectic fever, for the diurnal variation was from two to three degrees; but a second glance showed that the morning temperature was generally 96°, and the average between 97° and 98°. The original diagnosis in this case was tubercular peritonitis, and I must admit that when I saw the lad, early in May, this diagnosis seemed to me the most probable. The belly evidently contained a large quantity of fluid which did not reach the back of the right hypochondrium—either because of adhesion and matting of parts shutting it out, or because it was contained within the capsule of a cyst. The fact that the boy had lost flesh rapidly, and the peculiar character of the temperature chart to which I have above alluded, seemed to me to favour the view that the fluid was encapsuled by adhesions, the result of tubercular peritonitis. But my colleague, Dr. Abercrombie, came ultimately to the conclusion—and he had done so before he asked me to see the case—that the fluid was in a cyst, and that the cyst had developed from the left kidney. In other words, Dr. Abercrombie's view was that the boy had a left hydronephrosis, due in all probability to some congenital condition, which had led to very slow distension of the pelvis and destruction of the kidney substance. Such causes as a congenitally small ureter, an oblique entry of the ureter into the pelvis, compression of the ureter by an abnormal artery or by some band, have not uncommonly led to hydronephrosis, sometimes of enormous size. I thought that the case was not one of hydronephrosis, because the left side was little if at all fuller than the right. The swelling did not markedly distend the left, and could not be pushed back into the loin. Lastly, no one had succeeded in finding any indication of the colon on the front of the kidney. Had we practised inflation of the colon—an aid to diagnosis not sufficiently used—it is probable that the colon would have been demonstrated in its normal position, *behind* the cyst; and it would then have been pretty plain that some central cyst was present. So, with an open mind, on May 7th I operated on this boy. I made an incision just below the umbilicus, so that if the case proved to be a left hydronephrosis I could easily extend it upwards to reach the pedicle. On opening the peritoneum I found that the fluid was not "encysted," but that it was contained in a large, thin-walled cyst of deep blue colour. This cyst was covered by omentum, and very little further examination showed that it occupied the cavity of the small bag of peritoneum which you will remember comes down in front of the transverse colon between the four layers of omentum. There we found this cyst, with the stomach in front and above, with the colon below and behind, and the great cyst coming out between them, pushing apart the anterior and posterior layers of omentum in that way. I split open the anterior layers of the omentum and tapped the cyst so as to get more room for manipulation, and to find out where the cyst originated. I show you some of the fluid

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collected; you see it is of a deep greenish-brown colour. It looked to me like fluid that I have drawn off from pancreatic cysts. We got away twelve pints of this fluid, which was all contained in this one cyst. There was no fluid free in the peritoneum. Having emptied the cyst, I had plenty of room for investigation. I passed my hand to the left kidney, which I found normal so far as touch could discover. The colon, too, was in its normal position, but evidently it had been kept pretty empty by the pressure which had been exerted by this cyst, and thus it failed to yield any resonance on the left side. The stomach was pushed upwards and was much flattened against the diaphragm. The right kidney also was normal. Under these circumstances the most likely source of the cyst was the pancreas. So I more widely opened the omentum and followed the cyst up to its root. It sprang from the front of the pancreas, practically in the mid-line. The aorta could be felt pulsating immediately behind its base. By dividing a little fat and connective tissue on its outer aspect it was easy to remove the cyst entire. It left no irregular or depressed surface on the pancreas. So whether it was actually connected with the pancreas or not I cannot say. We examined the fluid, and it contained nothing but unformed detritus and cholesterine crystals under the microscope. It was albuminous, and had no digestive power whatever. So there is no positive evidence that this cyst sprang from the pancreas; all that is certain is that it originated behind the peritoneum in front of the pancreas.

It is to be regretted that we did not inflate the stomach and colon in this case; in all probability this would have enabled us to suspect pretty strongly the site of origin of this cyst.

I have a couple of sections of this cyst wall under the microscope for you to see. At one part there was a low, flat thickening on the inner surface of the cyst, so that the whole looked something like the membranes with a miniature placenta. One section shows the thin part and the other the thick, but the structure is practically the same in both. There are a large number of vessels in the lining membrane; and it is easy to account for the colour of the fluid by supposing that hæmorrhage occurred from these vessels, and that cholesterine was present in quantity—as it obviously was.

Epithelioma of Tongue (far back), Floor of Mouth, and Jaw.

The next patient is a man æt. 68, rather small and spare, but generally sound. Two months before his admission here, on the 11th May, he began to feel pain behind the angle of the right jaw. Shortly afterwards two lumps appeared there and increased in size. The pain became continuous, though not severe at first; and it often shot up towards the ear in the direction which is so characteristic of all tongue pains. Somewhat later, whilst drinking some hot tea, he found his mouth was sore, and then discovered that he had an ulcer in the floor of his mouth, far back. He has been losing flesh for two months,—that is to say, during the whole time that he has noticed anything wrong. The present state was described as follows:—Small, healthy-looking, and strong; breath offensive, teeth dirty; he can hardly protrude his tongue at all. It is obviously fixed on the right side near the anterior pillar of the fauces. The tongue is more prominent on the dorsal aspect on this than on the left side, but the surface of the tongue was normal. In the furrow between the tongue and the right jaw there is a considerable, hard-based ulcer, extending from the side of the tongue beneath the edge, right round the floor of the mouth to the gum, over the gum, and a short way on to the cheek, so that the outer edge of the epitheliomatous ulcer was formed by the cheek mucous membrane; the ulcer ran back to the root of the coronoid process, and came forward one inch from that point. Round about it the cheek mucous membrane was reddened and faintly granular for perhaps half an inch, but not thickened; I thought that was due simply to irritation. During the time he was here before operation this redness subsided somewhat. The jawbone was not thickened. The submaxillary glands formed a hard mass not bigger than a small walnut, which seemed adherent to the tongue. Examining bimanually, with fingers inside and outside the mouth, the growth did not seem to reach the mid-line, and I hoped that removal of one half of the tongue would be sufficient. The upper carotid gland appeared to be somewhat large; but really the extent of the glandular affection was very slight considering what a large epithelioma he had in his mouth. To summarise: the growth involved the right half

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of the tongue, far back, extending well down towards the hyoid bone; it involved the floor of the mouth, the gum and alveolar process, the mucosa of the cheek to some extent, the submaxillary lymphatic glands, and possibly the upper carotid. In spite of his age, having in view the fact that he seemed to be sound and a good healing type of subject, active and wiry, and having in view also the small amount of glandular affection, I thought it right to advise him to undergo a radical operation. This was performed yesterday, and consisted in the following. Before operation we endeavoured to carefully cleanse the mouth—a most important point—by scaling the teeth and by frequent washing out with boracic lotion.

The first stage of the operation was extra-buccal: a large Kocher's flap was raised on the right side, and all the connective tissue and lymphatic glands in the submental and submaxillary regions were cleanly removed, also the submaxillary salivary gland. This involved ligature of the facial vessels. Next the posterior belly of the digastric was defined and raised, and the connective tissue and lymphatic glands lying over and extending upwards from the bifurcation of the carotid was removed, although there was no clear sign of involvement of the glands. The last act of this stage was the ligature of the right lingual—difficult in this instance, because the space was very small, and several veins about the hypoglossal nerve bled annoyingly.

The second stage of the operation consisted of the steps necessary to provide free access to the primary growth. This was known to involve the jaw, and it was therefore imperative to remove the infected bone. But apart from this I can strongly recommend division of the jaw in front of the masseter (with subsequent wiring) as an excellent means of obtaining access to cancers of the root of the tongue and their extensions. This was, I believe, a favourite method with Langenbeck; but he divided the jaw through a cut made downwards from the angle of the mouth. The result of this must have been persistent paralysis of the lower lip on that side. This defect is easily avoided by cutting through the lip and chin in the mid-line in front, and connecting this incision with the Kocher's incision below the jaw. A large flap of the cheek and submaxillary skin can now be quickly raised, bleeding being much reduced by the previous ligature of the facial. Epitheliomata can now be cut out from the cheek just as if they were on the skin surface. This was done in the present instance, with a half-inch healthy margin. Then a chain-saw was passed internal to the jaw, the bone was divided in front of the growth, and the fragments were forcibly separated.

The removal of the growth constituted the third stage. The hinder fragment was sawn through for half its depth vertically in front of the coronoid process, well behind the ulcer, and the alveolar half was separated from the base with bone forceps. Next the tongue was split along the middle down to the hyoid bone, and the left half was drawn out through the gap in the jaw and removed in one piece, together with the right half of the floor of the mouth, the upper half of the jawbone to which the growth was adherent, and a piece of the cheek mucosa.

The final stage is that of repair. First the fragments of the jaw were wired together and the arch thus preserved. The next step, which I consider very important, was to sew together the cut edges of the mucous membrane, so as to cover as far as possible the raw surfaces which would otherwise discharge decomposing pus into the mouth and threaten the patient with septic broncho-pneumonia. During the whole of the operation every care was taken to prevent the entry of blood into the air-passages—another very important point, because if the blood gets into the bronchi and undergoes decomposition, septic broncho-pneumonia is a probable result. Finally, I like to paint all raw or sutured surfaces in the mouth with Whitehead's varnish.

With regard to *after-treatment*, the following are the most important points in these mouth cases:—First, sit the patient up as soon as possible. In the case of this patient the operation was finished at seven o'clock, and by ten o'clock he was sitting up in bed. As soon as you think that the shock has passed off, sit the patient up with the head somewhat flexed, so that anything in the mouth tends to drain out of it. In this position a patient breathes better, coughs and spits more effectively, and can be more easily assisted to get rid of mucus, etc. Next, when you have opened the mouth into the neck wound, see that drainage is as perfect as possible. You must be careful that there is no

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bagging in the neck, otherwise septic cellulitis is very likely to arise, and is very fatal; I think that drainage is best effected by an iodoform gauze petticoat and plug. Next keep the mouth clean. Twice a day we go over this mouth, brush the teeth with a dab in forceps, and clean away all mucus with dilute soda solution, especially from the floor of the mouth, and once a day, after such cleansing and drying, we paint the whole surface with Whitehead's varnish. He washes his mouth out frequently with boracic lotion, and always before and after food. We feed him *per rectum* until we find that he can take sufficient by the mouth; and this is sometimes early, sometimes late. As we increase feeding by the mouth we decrease the rectal feeding, until finally the latter is discontinued altogether. Feeding by the mouth can generally be best carried out by means of a feeder with a long rubber tube attached, which conveys the fluid almost to the point where reflex swallowing begins. If that is difficult, there will be no trouble in passing a soft tube down the œsophagus, and thus giving a good feed of milk and egg once or twice a day.

Although these operations are difficult, it is even more difficult to keep the patient alive afterwards. Where you take away large portions of the tongue and jaw, and open up the neck widely, the mortality is very high. Now what is the mortality due to? With regard to shock, in my experience, that is comparatively slight. Here is a man aged nearly seventy; but, although the operation lasted nearly two hours, the amount of shock was very slight indeed. Next hæmorrhage; of course in an operation like this every care is taken to prevent hæmorrhage, and the immediate and remote troubles resulting from it. With regard to secondary hæmorrhage, if you have a vessel of the size of the external carotid tied in a septic wound, there is considerable danger of secondary hæmorrhage. In this particular patient there is some danger of secondary hæmorrhage, for I placed a ligature on the very origin of the facial. Not long ago I lost a patient from bursting of his internal carotid just above its origin. Here the artery was a little dilated and its coats were very thin; it had been laid bare in the removal of glands, and three or four days after the operation the dilatation burst, blood entered the larynx, and the patient died, asphyxiated. Secondary hæmorrhage has often occurred after septic ligature of the external carotid; but I have not seen it after ligature of the primary branches of the external carotid. The next and most fatal trouble is septic broncho-pneumonia. That is caused by the inhalation of decomposing discharges from the mouth into the bronchi. The more nearly your operation approaches the glottis, the greater the tendency for these discharges to accumulate about the orifices and to be inhaled. We endeavour to guard against this danger by carefully controlling bleeding during the operation, by position, by sutures applied to reduce raw surfaces in the mouth to a minimum, by free drainage, by great local cleanliness, and the use of iodoform. Then there is local sepsis—septic cellulitis in the neck,—a very serious complication indeed, complicated as a rule by a large measure of septicæmia or pyæmia. Of course these septic wounds may be complicated by the ordinary diseases of wounds, such as septicæmia and erysipelas; but those which I have just mentioned are the most common causes of death.

Fibroma Uteri; Hysterectomy.

The next patient is one who had this 5-pound fibroma of the uterus which I show you growing rapidly, and causing profuse menorrhagia. She was only thirty-seven years of age. For the last year her periods have lasted a fortnight, with a fortnight interval, and at each period she lost very freely. She was in very good general condition, but the fibroma was increasing rapidly. This tumour occupied the hypogastrium, and reached to two inches above the umbilicus. It was smooth on the surface. I could feel nothing of the round ligaments, and no irregularity which indicated where the fundus of the uterus was. The cervix was soft, and looked in the natural direction, backwards and downwards. The sound entered upwards, backwards, and to the left. The notes say it shows a greatly dilated cavity. The operation was an easy one; both fornices were free, the whole mass was movable, and there was nothing to do but to tie the ovarian and uterine vessels on each side, remove the growth by supra-vaginal amputation, and close the deep and superficial wounds. Recovery, as you see, has been uneventful.

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Chronic Obstruction; Transverse Colotomy. Removal of Cancer of Splenic Flexure and Part of Transverse Colon.

The next case is a rather remarkable one. She is æt. 47, and her history is that she was quite well at Christmas, doing her work and not noticing any loss of flesh. About Christmas she suffered a little from constipation accompanied by some pain. For this she had to take a dose of liquorice powder once a week. On January 11th of this year she took half a drachm of liquorice powder, but it did not act. On the following morning she took a drachm. On the evening of the 12th she had a fluid motion, which was passed after much pain in the abdomen and colic. But after its passage she felt better, and she had a fair night. On the following morning, the 13th, she had three fluid motions, and so much pain in the abdomen that her husband sent for a doctor. He told her she had inflammation of the bowels. Her abdomen was somewhat distended, and he gave her a daily enema to keep the bowels open, and fed her by nutrient enemata, keeping her off things by the mouth except champagne and brandy. In three weeks the bowels were acting naturally, and she got downstairs. But some accident happened to the boiler, and as a result of standing about in the wet she got a chill, as she thought, and the very day she came downstairs she had to go back to bed again, with all her old symptoms—abdominal pain, constipation,—and these got much worse. This brings us on to February. From that time onward the history is one of chronic obstruction. The doctor did his best by means of enemata to get the bowels to act. Sometimes he succeeded and sometimes he did not. She began to vomit, and the vomiting became more frequent. After a time only champagne was kept down. The abdomen was constantly the seat of uneasiness, frequently the seat of colicky pain, and it was getting more and more distended. She was losing flesh rapidly all the time. It was just the ordinary history of intestinal obstruction.

Now increasing constipation and colic are symptoms which one must look upon with great suspicion in anybody of this woman's age. She was sent up under my colleague, Dr. Abercrombie, when I was away in April, and Mr. Gibbs saw her late at night. He found the abdomen very distended, and the patient very ill and feeble. She had a median hypogastric scar, and stated that her ovaries had been removed some years ago for dysmenorrhœa. He opened the abdomen, and found the bowels greatly distended, and tending strongly to protrude in quantity. He searched as well as he could under the circumstances, but without discovering the cause of the obstruction. He found a tense band of omentum passing down to something unknown in the left iliac region, and possibly compressing a piece of bowel; he divided this but, the result being unsatisfactory, and there being no time to waste, he took a piece of distended large intestine fixed it in the median abdominal incision, and drained it with a Paul's tube. The patient recovered from this operation, and when I came back to duty in May I found her thin and feeble, with a central anus below the umbilicus. The bowels were not acting quite satisfactorily, and the abdomen was too full for such a very thin patient. The anus presented a single opening. A finger introduced into it passed downwards and to the left easily; but a spur was found above and to the right, fitting closely over the orifice of the bowel in this direction, and suggesting that the bowel on the right of the anus was *below* it. Enemata had been given through the artificial anus, and observation of the result had led to the conviction that the fæces did escape from the bowel below and to the left of the opening. It was therefore thought that the loop of gut which was opened was twisted when it was fixed in the wound, so that its upper and right limb lay below and to the left of the anus. A further point was that it was uncertain whether it was the transverse or the sigmoid colon which had been opened.

On the other hand, it was clear that the woman had some obstruction of the large gut between the transverse colon and the anus, and it was probable that this was carcinomatous. Whether malignant or simple it was possibly removable, and the artificial anus could then perhaps be closed. A very thorough bimanual examination made me pretty sure that there was no growth in the rectum or at the pelvic brim. I thought I could feel something like a tumour beneath the left lower ribs, and the patient was somewhat tender here. Under these circumstances I obtained the woman's consent to a further operation, having for its object the discovery and, if possible, the removal of the obstruction and the closure of the artificial anus. I

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opened the abdomen in the mid-line, circumscribing the anus with the knife, freeing the bowel and drawing it well out, the anus being guarded to prevent soiling of the wound. It proved to be the transverse colon. It was not twisted on itself. The piece of gut into which the anus led most easily lay below the anus; nevertheless it was quite full of fæces, and no doubt did extrude some under the stimulus of an enema. The closure of the opening of the upper piece of bowel by the spur was apparently due to some accident in suturing. It probably did form a slight obstacle to the passage of fæces and flatus, and prevented the abdomen from collapsing fully. I found that the splenic flexure was the seat of a large carcinoma, fixed, like the flexure itself, by its peritoneal connections, but not infiltrating, and apparently it had not spread to any glands. It was therefore fit for removal. The artificial anus lay some nine or ten inches above and to the right of the growth, and it seemed to me that the quickest and best thing I could do for the patient would be to excise the growth, the artificial anus, and the intervening colon, and then to join the cut end of the transverse colon to that of the descending colon. This was done without difficulty, and for the sake of greater speed I used a Murphy's button to effect the intestinal junction. The abdomen was cleaned in the ordinary way, and the wound was closed.

Although the patient has not had quite an uneventful time since, I think she is doing fairly well. I was anxious about her the next day because she had a good deal of pain about the button and some fever, and I feared she might be getting peritonitis or a localised abscess. The temperature, however, quickly subsided, as did the pain also; and the general condition remained good. About the fourth day I found the left loin red and œdematous, and next morning I made a small posterior opening through the muscles and evacuated a little fœtid pus. Since then she has gained ground steadily, though she still has a sinus in the loin, from which a little sweet pus is discharged. There has been no escape of fæces or flatus. The Murphy's button came away unknown to the patient on the twelfth day. She was fed chiefly by rectum at first, and the enema of boracic lotion given daily to wash out the rectum very soon provoked the passage of quite considerable motions. Indeed, but for the abscess recovery would have been singularly easy.

The chief lesson to be learnt from this case is that a patient presenting symptoms of chronic obstruction should have the abdomen explored long before she reaches such a pass as this woman had arrived at.

Note.—In December, 1902, Mr. Gibbs saw this patient. She was well nourished, doing her work, and, so far as he could discover, free from recurrence.

THE RUPTURE OF ABDOMINAL ANEURYSMS.*

By LEE DICKINSON, M.D.Cantab., F.R.C.P.,
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ANEURYSMS of the abdominal aorta, or what is much the same thing, aneurysms not purely aortic, but involving the cœliac axis or the origin of the superior mesenteric artery, in the neighbourhood of which most of them are situated, are far more prone to end in rupture than thoracic aneurysms. An insignificant minority become cured, and scarcely more destroy life by pressure upon various parts; the almost invariable end is rupture. A special interest therefore attaches to the manner and place of rupture, and to this part of the subject I will confine my remarks.

The abdominal aorta lies immediately behind the peritoneum, and, as was only to be expected, it is here that the aneurysms most frequently rupture. Considerably more than half rupture, in the first place at any rate, behind the peritoneum. In such cases the extravasation generally takes place into the loose and capacious cellular tissue of the lumbar and iliac regions upon one side of the spine, where the mischief is often limited, however temporarily, by coagulation of the blood and the formation of a false sac—a so-called diffuse or traumatic aneurysm. Under less favourable circumstances, especially in the case of aneurysms involving the cœliac or mesenteric arteries, the extravasation is bilateral from the first; and if the blood is poured out in overwhelming volume, it may spread without limit beneath the peritoneum in all directions.

Even the most extensive subperitoneal hæmorrhage is not suddenly fatal. The general symptoms of internal hæmorrhage may be overshadowed by the agonising pain that attends the separation and stretching of the peritoneum. For instance, a case became the subject of consultation at St. George's in 1893 on account of the sudden onset of severe abdominal pain vaguely distributed behind and around the abdomen. Aneurysm was not known to exist, and the diagnosis was at first quite doubtful; but I remember a shrewd observation of Mr. Clinton Dent that the severity of the

* Part of a clinical lecture delivered at St. George's Hospital on February 2nd, 1903.

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pain was a point in favour of retro-peritoneal hæmorrhage. The patient died some hours later, and the post-mortem revealed a small ruptured abdominal aneurysm and an enormous collection of recently extravasated blood behind the tensely stretched peritoneum, from the diaphragm to the pelvis on both sides of the spine.

A diffuse aneurysm in the cellular tissue of the lumbar and iliac regions of one side is compatible with life for days or even months, and is tolerably familiar, clinically, as a vast, deep-seated, fluctuating tumour, generally on the left side, which, but for its sudden appearance and the presence of pulsation, might be mistaken for an abscess. We have these cases from time to time in the hospital; in fact, almost as often as abdominal aneurysms pure and simple. The most striking case of recent years was that of a middle-aged man who was under the care of Dr. Penrose in the summer of 1897. He was admitted with an indefinite fulness in the right hypochondrium, which, during the three months and more that he was in the hospital, grew into a definite pulsating tumour presenting in the iliac region. This tumour proved to be formed by a vast false aneurysm behind the peritoneum, reaching from the liver to the groin. Death was due to gradual exhaustion, without any sudden rupture from first to last, in which respect, as well as in its chronicity, the case was exceptional. As a rule, these diffuse aneurysms are acute in their onset, and much shorter in duration than in the case just quoted; but, however chronic, they always tend to progressive enlargement, and sooner or later prove fatal. A common ending is secondary rupture into the peritoneum or pleura. External rupture does not occur. The psoas and iliacus muscles may be infiltrated with blood, and external pulsation may be both violent and extensive, but for the blood even to make its way so near the surface as to cause discolouration of the skin is practically unknown.

Somewhat less than 50 per cent. of abdominal aneurysms rupture directly or indirectly into the peritoneal cavity. The ordinary result of this is fulminating hæmorrhage; there may be a momentary pang from the rent of the peritoneum, but death is sudden. There is reason to believe, however, that rupture through the peritoneum is not always suddenly fatal. Stokes, in his 'Diseases of the Heart and Aorta'—a work which, for circumstancial interest and clinical detail, has never been surpassed,—relates a case of what was supposed to be "hæmorrhage by successive gushes" into the peritoneal cavity, the patient for three days before his death having had each day a fainting fit. The exact place of rupture is not mentioned, but "the aneurysm lay under the left lobe of the liver, depressing the pancreas"—that is, behind the cavity of the lesser omentum. It is possible that hæmorrhage into this comparatively small cavity, from which the only outlet is the narrow foramen of Winslow, may be less rapidly fatal than hæmorrhage into the general cavity of the peritoneum; but this is a suggestion that rests upon probability rather than proof. In a case which occurred at St. George's in 1894 an aneurysm in the position of the cœliac axis was clearly shown at the post-mortem to have ruptured into the cavity of the lesser omentum, whence the blood had escaped by the foramen of Winslow. The patient died suddenly after an attack of vomiting, but the order of events is not clear from the clinical history; at any rate, there was no preliminary fainting attack. Rupture into the lesser sac is certainly exceptional; the pressure of an aneurysm in this situation tends to cause adhesion of the omental layers, the sac becomes obliterated, and the rupture takes place into the general cavity of the peritoneum.

Rupture into the alimentary canal is rare. The stomach escapes owing to its mobility. Not so the duodenum, which of all the intestinal tract is the most likely place, and after this the descending colon. As a rule, rupture into the bowel is rapidly fatal by hæmorrhage, in which respect it contrasts with the often gradual leakage of thoracic aneurysms upon the mucous surface of the air-passages. In the only duodenal case with which I have been connected, the rupture was signalised by profuse hæmatemesis and almost sudden death. Post-mortem the small intestine was loaded with blood.

According to Sibson, who analysed 177 cases, and so was fairly entitled to speak in percentages, more than 15 per cent. rupture upwards into one of the pleural cavities; mainly the left. Stokes gives a minute description of eleven cases of abdominal aneurysm, no less than five of which ended by rupture into the left pleura. This was no doubt an accidentally high proportion; but still,

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rupture into the left pleura, either direct or indirect, by way of the retro-peritoneal tissue, is one of the commonest ends. The slightly leftward inclination of the aorta as it passes through the diaphragm is one reason for this prevalence of rupture into the left pleura as compared with the right, but probably the chief reason lies in the structure and disposition of the diaphragm itself. The left wing is naturally weaker than the right, if one may judge from its far greater liability to congenital deficiencies and hernia; moreover it lacks the protection which is afforded to the right wing by the liver. Both wings present a *pars minoris resistentiæ* through which not only blood, but inflammatory and gaseous effusions in the retro-peritoneal tissue are apt to break upwards into the pleura. According to Dr. H. Curschmann, of Leipsic,* whose paper upon the relations of the retro-peritoneal cellular tissue is of great anatomical and clinical value, this weak spot or space is in rather than behind the diaphragm, for it is in front of the transversalis fascia and ligamentum arcuatum externum. He describes it as triangular in shape, the base being formed by the twelfth rib, the apex by the central tendon, the sides by the crus internum and crus externum; the roof is formed by the pleura, and the floor merely by a continuation of the retro-renal connective tissue. To some extent the front part of this space is guarded by the kidney, but this displaceable organ can be little protection against the pressure of a growing aneurysm, and still less against that of a violent effusion of blood; thus it happens that rupture into the pleura takes place.

As a result of this accident, death is often sudden. The patient is subjected to copious hæmorrhage, amounting perhaps to three or four pints, and simultaneously to compression of the corresponding lung and displacement of the heart—a concurrence of events which is apt to prove fatal within a few minutes, or at most within a few hours. Sometimes, however, the hæmorrhage into the pleura is gradual or intermittent, either because the cavity is guarded by adhesions that do not at once give way, or because the rent in the membrane is not at first a free one, and death may be delayed for days. Stokes draws particular attention to hæmorrhage by successive gushes into the pleura, which is a matter of actual occurrence, and one that may give rise to practical questions of diagnosis and treatment.

I am not aware whether tapping the pleura has ever been practised under the circumstances, nor whether the operation would be for good or for evil; but I have been concerned in a case where it was within measurable distance of being undertaken. In this case the aneurysm, which came from the highest part of the abdominal aorta on its posterior surface, was beyond the reach of physical diagnosis; the patient was under surgical care for supposed caries of the spine, which disease was simulated by dorsal pain and spastic gait due to erosion of the vertebræ and pressure on the nerves by the aneurysm. When a fluctuating non-pulsatile tumour developed in the left loin, together with signs of fluid in the pleura above, the condition had all the appearance of a descending empyema in connection with the spinal disease, and the propriety of operation was seriously considered. The man lived four days longer without operation, and the post-mortem confirmed the suspicion, which had already been aroused, that the true state of affairs was a ruptured abdominal aneurysm and a large effusion of blood into the pleura.

It is far less common for the blood to travel upwards through what are generally supposed to be weak spots in the diaphragm—the aortic and œsophageal openings,—and thence into the posterior mediastinum and roots of the lungs. The late Dr. Bristowe recorded one such case, the main result of which was that the cardiac end of the œsophagus became encircled by a ring of coagulated blood, and the patient died, not from hæmorrhage, but from œsophageal obstruction. In a case which occurred at St. George's in 1889 a considerable quantity of blood collected in the mediastinum and passed into the root of the right lung; but since the larger collection was in the pleura, it is probable that the diaphragm had been penetrated in the usual way—that is, through the space described by Curschmann.

A few curious cases have been described of rupture into the vena cava, the spinal canal, and the pelvis of the kidney; but none such have occurred within recent years at St. George's, the experience of which hospital, comprising examples of all the more ordinary kinds of rupture of abdominal aneurysms, I have taken for the foundation of this lecture.

* "Topographish-klinisch Studien," 'Deut. Archiv f. klin. Med.,' 1894, p. 53.

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CLINICAL LECTURES
ON
DISEASES OF CHILDREN.

LECTURE V.

THE WASTING DISEASES OF INFANCY—MARASMUS AND CONGENITAL SYPHILIS.

Delivered at the London Hospital, November 4th, 1902.

By ROBERT HUTCHISON, M.D.Ed., M.R.C.P.,
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GENTLEMEN,—I wish to consider with you to-day *wasting* as a symptom of disease in infancy. When you go into practice it will occur to you almost every day to have a baby brought to you by its mother with the complaint that it is wasting. She will tell you, in the common phrase, that it was "a beautiful baby born," but that, either from its birth or at some period subsequent to its birth, it began to waste. And very commonly you will find on inquiry that it began to waste when artificial feeding was adopted. I would point out to you that wasting is not a disease in itself; it is, strictly speaking, a symptom, not a disease. So when a child is brought to you with that complaint it is your duty to try to find out to what the wasting in that particular case is due; and there are many possibilities which must present themselves to your mind.

The first is that *the feeding may be insufficient.* That is the first possible cause of wasting, and, of course, a most natural one. If the case be one of a breast-fed child you may find on analysis of the milk that it is poor in fat, or that the total quantity secreted is small. If it be a bottle-fed child you will find that by increasing the strength of the mixture on which the child is fed it will start to gain weight. So there is no great difficulty in diagnosing wasting from mere insufficiency of nourishment.

Then you may have, as a second cause of wasting, *inability on the part of the child to suck.* That is a cause which you are more likely to overlook. The child may be unable to suck the breast or the bottle because of local conditions; I mean such things as stomatitis, an inflamed and painful condition of the mouth, which makes the movements of the tongue in sucking productive of acute pain. In such a case the child will cease to suck, and whilst the local condition lasts it will waste. But that, as you will see, is not likely to be a case of chronic or prolonged wasting, because those local conditions in the mouth are, fortunately, usually of only short duration. Then, a child may be unable to suck because it is unable to breathe. I mean that if the nose is blocked up the child has only one channel left by which both to breathe and to feed, and in that case it is unable to breathe whilst it is sucking; and owing to that it is probably unable to get down a sufficiency of nourishment, and so again it wastes. Other local causes are such conditions as hare-lip or cleft palate. I have known children waste severely on account of their inability to suck from the presence of a cleft palate.

In the third place I would remind you again of the possibility, in the case of artificial or bottle-fed children, that *the teat may be unsuitable.* I saw a child not long ago, for instance, which was brought to me when a month old and weighed only 6 lbs. 7 ozs. At first I could find no obvious cause for its wasting. We made a little modification in the feeding, and it was brought a week later weighing 6 lbs. 5 ozs. Still we could find no explanation for the loss of weight. It came back a week later weighing 6 lbs. 4 ozs. Then I looked at the teat, and found it was of the "leech-bite" form, which, as I told you before, often prevents the milk coming through easily. I recommended that the bottle should have a new teat, which was done, and the child was brought back the next week weighing 6 lbs. 10 ozs., and in a fortnight it weighed 7 lbs. 12 ozs. That is an example of how real this cause of wasting may be, no matter how trivial it may appear.

In the fourth place, an infant may waste on account of *persistent indigestion;* that is to say, if a child is getting a mixture which is too strong for it, a mixture which is chemically quite sufficient to supply its nutritive requirements but which is too strong for the digestive organs, that child will waste, with the ordinary symptoms which I described to you in the last lecture—namely, colic, vomiting, and probably occasional diarrhœa. So that the treatment of this form of wasting will resolve itself practically into treatment of the disorders I described to you when we last met.

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A fifth cause, and one which you must never overlook, is the possibility of *the existence of organic disease.* I refer particularly to congenital syphilis, to latent tuberculosis in some form or another, and, though this is rarer, to the presence of an undiscovered empyema. All those forms of organic disease may be the cause of serious wasting, and I shall consider them separately in a few minutes.

We come now to the last group of cases in which one finds wasting, those, namely, in which there is *no apparent or discoverable cause.* It is to that group, a comparatively small one, that the term marasmus, or, as it is sometimes called, athrepsia, is strictly applicable. I do not think one has any right to talk of marasmus when one is referring simply to a case of insufficient feeding, or where one is dealing with persistent indigestion, or where there is an organic cause, such as those I mentioned in the fifth group. I think one should reserve the term marasmus for those cases where there is wasting for which one can find no sufficient explanation. I saw yesterday afternoon in the post-mortem room an autopsy upon a baby which belonged to this group, and the house physician who brought the organs to me said he was puzzled at being unable to find any evident cause for the child's death. That is exactly what one expects in those cases; you search the body thoroughly after death, but can find no reason at all for the wasting and fatal issue. Naturally people have advanced theories to account for such an occurrence. There is one school who state that if you examine the alimentary canal histologically in those cases you will generally find it to be the seat of microscopic alterations; they assert that you will find maldevelopment of the lymph-follicles and Peyer's patches, and that you will find also atrophy and catarrh of the glands of Lieberkühn. On the other hand, it is denied strenuously by another group of people that these apparent microscopic findings have any significance at all; they say you will find them as commonly in children who are not wasted, simply as a result of post-mortem change, and if you inflate the bowel of children who have died from other diseases you will find a similar alteration brought about purely mechanically. There is perhaps more to be said for those who maintain that this condition of apparently causeless wasting is not due to anything on this side of the alimentary canal at all, but that it is a tissue vice, a failure of assimilative power on the part of the tissues generally, perhaps on the part of the liver particularly. I confess that that sounds rather vague, and I admit it is vague, but I do not know of any way in which to put it more clearly. And such a condition is not unthinkable. You can find analogies for it in certain diseases of grown-up persons. There is the failure of assimilative power with regard to carbohydrates in diabetes; you know of a similar failure as regards fat in some cases of obesity, where the fat is stored up instead of being consumed. There is also the failure of assimilative power with regard to proteids in some forms of gout. And it is possible that some children come into the world with congenital inability on the part of their cells to assimilate nutriment properly; and the conditions under which many marasmic children are born would conduce to that congenital feebleness. They are often born at the end of large families, when the reproductive power may be assumed to be on the wane or to be partially exhausted, and they have often been brought up under very unsuitable hygienic surroundings, and probably have been always badly fed.

So much for the pathology, if one may call it so, of this condition. The symptoms I need hardly describe to you. The chief one is persistent loss of weight, and in the group of cases which I am now particularly considering—those to which the term marasmus is strictly applicable—there need be no other symptom at all; no special disorder of digestion, no diarrhœa, and no vomiting. The child simply goes on losing weight, with varying rapidity; it becomes shrunken and emaciated, and finally presents that picture of extreme feebleness and atrophy which is so commonly seen in the out-patient room.

The *treatment* of true marasmus you will find by no means satisfactory. The treatment of wasting due to many of those other causes I have mentioned is quite easy; it is easy to increase the amount of food, or to alter it. It is often easy to correct indigestion, or even to deal with some forms of organic disease, but it is not easy to improve the assimilative power in one of those marasmic children. You will find that you exhaust all your ingenuity in devising mixtures of artificial foods without any success; and, indeed,

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these are the cases which are brought to you with the history that they have had all sorts of foods without any benefit. If you can, you will probably find that the best thing to do with those marasmic children, assuming that they are not being fed from the breast of the mother, is to get them a wet nurse. They certainly do better on human milk than on anything else, and you will save a certain proportion of them by having recourse to that means. Failing that, you often have to use the most easily digested mixtures which you can find. For instance, peptonised milk may be used, and by extreme care in feeding, and by husbanding the child's strength at the same time—that is to say, by keeping it very warm so that its expenditure of heat is comparatively small—you may sometimes be able to tide it on until the period when the first teeth are cut. Fortunately, about that period the digestive power of such children often appears to undergo a sudden alteration for the better, and towards the sixth month they begin to be able to deal, not perhaps with milk, which in these cases often presents a great difficulty, but they begin to be able to digest starchy foods, and thrive upon them; and after that all the difficulties which you have been combating may disappear. But you have to be prepared for frequent disappointment, inasmuch as these children often go persistently down-hill and die, no matter what you do for them. One peculiar symptom which you are likely to meet with towards the termination of these cases of marasmus is that the temperature, which has been previously normal, or perhaps subnormal, rushes up to a very high point. I have known it go up to 108°, and even higher. This development of hyperpyrexia in the last hours of life is a thing which occurs not uncommonly, and you may perhaps think, on that account, that there will be signs of tuberculosis after death, but you do not necessarily find such signs at all. I do not know what it means, but it is always a symptom of the very greatest significance. I have never known a case which developed it survive more than a few hours at the most.

I will now pass from the subject of marasmus to consider those two organic diseases, congenital syphilis and tuberculosis, which so often manifest themselves chiefly by the symptom of wasting. We shall take *congenital syphilis* first. I shall not deal at all exhaustively with it, and I shall not describe it as it occurs in older children. I want merely to deal with congenital syphilis as it occurs in young infants, and particularly to tell you how you are to recognise it; because upon its recognition, I need hardly say, the proper treatment of that group of cases in which the wasting is induced by the existence of the syphilitic taint must always depend.

In the first place, if you suspect that congenital syphilis may be the cause of wasting in a case with which you have to deal, you must inquire carefully into the family history. I have previously told you that you must inquire carefully into the family history of all infants, but you must particularly do so in those whom you suspect to be the subjects of this disease. You will ask about the health of the previous children; about the number of still-births, if any; whether there have been any miscarriages; and whether the child before you was carried to term. You may get a positive history which is extremely suspicious. You may have a narration of cases of still-birth or repeated miscarriages, or the present child may have been prematurely born. But you must be prepared every now and then to hear, in a case in which you may conclude on other grounds that the child is the subject of congenital syphilis, that the family history is good. In other words, you do not always get a suspicious family history, and you must not be led off the scent by its absence. What, then, are the signs by which you will recognise the existence of the syphilitic dyscrasia in a young child? You will often, by a mere casual inspection, be able to decide almost at once that the child is the subject of syphilis. There is the characteristic facies which has been often described. The child has a shrunken or wizened up appearance. The skin has a curious earthy tint, which has been compared to that of a mixture of coffee and milk, and the nose often shows a depression of the bridge. There is commonly also an absence or a scantiness of the eye-brows. When you look more carefully at the head you will find frequently, although by no means always, a peculiarity of the hair to which I want to direct your special attention. This peculiarity of the hair has never, so far as I know, been described in books on this subject, but you will often find it of very real service in diagnosing congenital syphilis. You will observe in many of these children that

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the hair is unusually long, that it is straight, fine in texture, and unusually dark in colour—a combination of things which one is in the habit of describing as the "syphilitic wig." The existence of this as a sign of congenital syphilis in early life was pointed out to me some years ago by Dr. W. S. Colman, and I have frequently found it of the very greatest value in diagnosing early cases of congenital syphilis. I shall show you immediately lantern slides illustrating examples of the syphilitic wig, in order that you may be able to recognise it afterwards.

You will find *signs in the bones* in many cases also; a bossing of the fronto-parietal eminences is a common, but not an invariable sign. The existence of those areas of softening behind the ears and along the occipital suture, to which the term cranio-tabes is applied, is frequently to be made out. You will often find also that there is a slight degree of hydrocephalus. Along with that you may observe a curious ridging of the edges of the sutures, and a slight thickening round the margin of the anterior fontanelle, the bone being, as it were, slightly heaped up. I do not say that that heaping up is invariably due to congenital syphilis; but, like head-bossing and cranio-tabes, I think it is more commonly due to that than to anything else. Then, passing down over the child, you will frequently find the existence of an *eruption;* but here, again, not necessarily always. Nor is the eruption of congenital syphilitic disease always easy to recognise. It occurs, as you probably know, more commonly about the nates than anywhere else. But you will remember that in that region also eruptions of the nature of intertrigo, due to irritation by the stools or by the urine, are not by any means uncommon, and you will not always find it easy to tell when you are dealing with a syphilitic eruption and when you are dealing with one which is due to this irritation. But there are certain points which will help you. One is the colour of the eruption. In congenital syphilis it has that coppery tint which is so characteristic of syphilitic skin lesions wherever found. You will find also that whereas an intertrigo rash appears only on the opposed surfaces, those which touch one another, a syphilitic eruption tends to wander down over the limbs, perhaps over the calves, and tends to appear on the soles of the feet or the palms of the hands. And when it appears in these situations it often has a silky or glistening look.

Snuffling is another common sign, as you know, but I would warn you not to attach too much importance to it. Snuffling and the presence of an eruption are signs which are apt to be overrated as a means of diagnosing syphilis; for those two signs in children are not by any means always due to that disease. Moreover many syphilitic children do not have snuffling at all. Other causes of it are the presence of a mere cold in the head, or the existence of adenoids. I shall have a good deal to say in a subsequent lecture upon the subject of adenoids in children; and I only want to point out now that adenoids do occur in infancy, and that they may give rise to snuffling and discharge from the nostrils which you may mistake for the signs of syphilis. A cold in the head you should not mistake for it, because the cold is usually of shorter duration, whereas the snuffling of congenital syphilis lasts for some time. The snuffling of syphilis does not begin immediately after birth; there is usually an interval of at least two or three weeks, and it is always accompanied, sooner or later, by the discharge of a yellowish irritating fluid from the nose.

There are certain other symptoms which you may look for. One is the existence of thickenings of the phalanges—what is called *syphilitic dactylitis*. You will find that of help in the diagnosis in a few cases; and I have also known cases recognised by the existence of gummatous deposits, particularly gummatous epididymitis, which may occur in quite young infants. You will perhaps get some help from an examination of the viscera. You may possibly find splenic enlargement. This occurs in about 50 per cent. of cases of congenital syphilis, but you must not expect to find it great in degree. A spleen which has been enlarged by congenital syphilis is not often very big; it is palpable, but usually not more than that. An affection which also occurs sometimes in this disease is an inflammation of the ends of the long bones, *syphilitic epiphysitis*. I have seen recently two cases in the out-patient room, one of which was shown at a meeting of our Medical Society. It is an affection of the ends of the bones at the point where ossification is going on, leading to enlargement and tenderness; and on account of the tenderness there is a loss of ability to use the limb,

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sometimes spoken of as syphilitic pseudo-paralysis, so called because it is not a genuine paralysis but an inability to make use of the limb from the existence of pain. I shall have more to say about that when I talk about paralysis in infancy generally.

Last, I would mention a symptom which has been emphasised by Dr. Eustace Smith, and which one sometimes finds of use in diagnosis, and that is *restlessness at night*. Syphilitic children, for some reason or other, are apt to be wakeful and restless at night, and to cry a great deal at that time. Dr. Smith suggests that this may be due to the existence of periosteal pains, just as there are often pains in the bones of adults who suffer from syphilis. At all events, you will sometimes find that this symptom will put you on the right track, and enable you to diagnose the existence of a syphilitic taint.

When all is said and done, and you have all these possible signs before you, there will still remain a residuum of cases in which you must be in doubt, because there are many children who waste merely because they have a slight syphilitic taint; they are not, strictly speaking, sufferers from congenital syphilis, but there is a syphilitic strain in the family, and there is enough of the poison in them to affect their nutrition adversely; and it is in these cases that one often has got to be guided in the diagnosis by the results of treatment, by giving antisyphilitic remedies and seeing what happens. You will remember I showed you some charts of wasting infants, in whom the administration of grey powder at once caused a gain in weight; and in a certain proportion of these cases that result is probably due to the existence of a congenital syphilitic taint which one has overlooked. I told you that it is not invariably due to that. I think one sees grey powder cause a gain in weight in cases in which one has every reason to exclude syphilis; but in some of the cases the explanation of such a rise in weight may be that the child has been slightly affected by this disease.

If you have satisfied yourself that wasting is due to congenital syphilis, there is a definite line of treatment marked out. You have to treat the syphilis by mercury, and the best way of doing so is by the mouth, giving half a grain of grey powder in this way four times a day. Begin with half a grain in the morning and half a grain at night, until you have got up to that dose four times a day. It is important to recognise signs of over-dosage. How shall you know when you are giving too much mercury? You know that you can recognise over-dosage in adults by the occurrence of salivation; but children never salivate with mercury. I do not know why that is, but it is probably due to the fact that the salivary glands do not develop well until the teeth are cut. At all events, salivation is not to be looked for when giving mercury to young children. The great sign in infants is the appearance of diarrhœa. So if during your treatment with mercury diarrhœa develops, remember it may be due to the giving of too large doses.

How long shall you continue to give mercury? That is a question which often presents real difficulties in practice. Many people advise you to stop giving mercury when all the symptoms have disappeared. I think those who recommend you to continue the treatment for nearly a year, in at least moderate doses, are probably better advised. There is a danger in stopping too soon. Over and over again it has been my experience to stop mercury when everything seemed to be going well, and to have the child brought back a month or so later with definite signs of syphilis again. Of course you often find it difficult to persuade mothers to allow treatment to be carried on. The tendency in hospital practice is for patients to cease attending when the symptoms have disappeared, so you must impress upon parents at the outset the necessity for prolonged treatment. Sometimes you will find it difficult to give mercury by the mouth; there are some children in whom even quite small doses produce diarrhœa, and in these cases inunction into the skin is a good method of treatment. One generally uses blue ointment, about half a drachm of it being rubbed into the abdomen once, or at most twice, in the day, after which the skin is covered by a flannel binder, so that gradual absorption of the ointment goes on. You will find syphilitic children tend to suffer from a sort of cachexia, and from anæmia, and if that is a prominent symptom it is advisable to give cod-liver oil and iron as well as mercury. Indeed, there is reason to suppose that the prolonged administration of mercury is in itself apt to produce anæmia, and that is probably why many people advise its early discontinuance. So when

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you go on giving mercury after the symptoms have disappeared, you may find it well to give iron and cod-liver oil in addition.

What about the treatment of local lesions? I think you should treat the snuffles by syringing out the nose with a solution of borax and putting a weak mercurial ointment up the nostrils by means of a feather. That prevents the crusts from drying and the nose from becoming blocked up. I usually employ weak white precipitate ointment for the purpose. The other chief local symptom which one has to treat is the eruption, and that you can do either by weak corrosive sublimate baths or by dusting powders containing calomel. Sometimes you may use a combination of weak white precipitate ointment and zinc ointment.

I will now show you some slides, and next time we will begin the consideration of tuberculosis as a cause of wasting.

TREATMENT OF RICKETS.

DR. BURNEY YEO.

In hand-fed children it is most important to see that they are not brought up on foods consisting chiefly of carbohydrates, as is so commonly the case with the poor. The milk they get should be good in quality and sufficient in quantity, and should contain its full proportion of cream, or even more than this. In winter a little cod-liver oil, made into an emulsion with lime water and sweetened with milk sugar, is an excellent addition. Each teaspoonful should contain about 10 to 15 minims of the oil, and we must see that it does not disagree with the child's stomach. Raw meat juice is a most valuable addition to the diet, as it is rich in salts. A little phosphate of lime may be added to the milk taken. Raw meat pulp has also been administered with great advantage in these cases, even up to two ounces daily, according to the age of the child. For older children milk puddings are suitable, made with "entire wheat flour." If the child has been suckled by a delicate and exhausted mother it should be at once weaned and a diet prescribed such as we have just indicated. We repeat that farinaceous foods are quite inadequate alone for the nutrition of such children; they are not only poor in lime salts, but they are prone to undergo lactic acid fermentation, which is calculated to aggravate the disease. In older children sandwiches of raw meat pulp, meat juices and broths, eggs beaten up with milk or lightly boiled, and the juice of fresh fruits may be added to the diet from time to time. We must not overdo the giving of fat, nor make too sudden changes in the child's diet, as digestive disturbances are easily set up. Fresh air and plenty of sunshine in a healthy country district or at the seaside, and removal from close, dark, overcrowded city dwellings, is of the greatest importance. Good ventilation of sleeping and dwelling apartments should be seen to. The air of the seaside will be found of special value in many cases. Bathing and sponging with tepid water night and morning is useful; the child should be dried quickly, and careful friction with the hand applied. A little olive oil or cod-liver oil may be rubbed in at the same time. Children who suffer from this disease are very sensitive to chill and apt to fall victims to broncho-pneumonia, pulmonary collapse, etc., and they must therefore be warmly clad in well-fitting woollen clothing, and a flannel band should be worn round the abdomen. But the garments must fit easily, so as not to hamper the respiratory movements or those of the limbs.

The above is taken from the new and revised edition of the widely-known 'Manual of Medical Treatment,' by Dr. Burney Yeo, published by Messrs. Cassell & Co. It is a pleasure to congratulate the author on the success of this book, and it is equally a pleasure to read in the preface of this new and revised edition of how he has received from all parts of the empire the most flattering acknowledgments of its value and usefulness. The author has not only rendered good service to medicine, but he has also set a high standard of excellence for medical writers, and it is to be devoutly hoped that in the latter respect his example will find a wide sphere of influence. The additions in this edition comprise the consideration of hay fever, of erysipelas, of cerebro-spinal fever, and of rickets, scurvy, and purpura. These subjects are very ably dealt with, and serve to indicate the great amount of labour which this work has entailed on the author.

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A DEMONSTRATION OF SURGICAL CASES

(TREATMENT OF TUBERCULOUS JOINTS)

At the Hospital for Sick Children, Great Ormond Street, W.C.

By F. J. STEWARD, M.S., F.R.C.S.,

Assistant Surgeon to the Hospital and to Guy's Hospital.

I THOUGHT our talk this afternoon would be better in the nature of a demonstration than anything else, and accordingly I asked a number of patients to come up to be shown to you. I think the best plan will be to have the cases in and show them to you first, and then go over with you the main points which they serve to illustrate.

CASE 1. *Tubercle of Knee-joint. Arthrectomy.*—This little girl first came here in the early part of the summer of 1900. She then had the symptoms of well-marked tuberculous disease of the knee-joint. This was treated by the repeated application of plaster of Paris. But no improvement took place; on the other hand the knee got more swollen, so she was taken in and an exploratory operation was done. It was not done by me, so I cannot tell you much about it, except that apparently she afterwards relapsed, and I think part of the wound became septic, and she was left with a sinus. I saw her in August, 1900, when the sinus was still open. She was admitted to the hospital, and I decided to operate again. I made a large incision across the front of the joint and explored it. I found that the whole of the joint was full of granulation tissue, and there was much bone disease. In fact, the note which I have is that in the lower end of the femur there was a hole into which I could put my index finger—an osteomyelitis of the lower end of the femur. I removed the whole of the synovial membrane carefully with scissors, and then I scraped out the cavity in the bone and plugged it with small pieces of decalcified bone and iodoform. The sinus was excised, and

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the whole of the rest of the wound was sewn up. She did far better than I could have hoped she would, and when she left the hospital I treated her with cod-liver oil and the usual drugs, and kept the limb in plaster. I kept her in plaster of Paris till the beginning of this year, eighteen months after the operation. Her knee is sound, and she has got fair movement in it, and can run and walk quite well. She can flex it almost to a right angle, and can get it quite straight. Another point to note is that there is no shortening of the limb. She does not seem disposed to do much now, but she is very energetic at times.

Case 2. *Tubercle of Elbow-joint.*—Here is a little girl who came up first when she was two years of age, and now she is four. She had well-marked disease of her elbow-joint when she came in October, 1900, and she had then had the disease five months. There was a good deal of swelling about the outer side of the radius; no pronation or supination could be performed at all, and her elbow was fixed in the position I show you. I applied first of all some mercurial ointment and an internal angular splint, which was kept on from October to the end of November. A good deal of her swelling having subsided and movement showing evidence of returning, I put on plaster of Paris, with the forearm at about a right angle. That was at the end of November, 1900. She continued to wear the plaster of Paris and improved steadily the whole time, and the plaster was removed finally a very short time ago, namely, on April 2nd, and since then she has been allowed to use her arm. The elbow can now be almost fully extended, and can be flexed well beyond a right angle. You will also notice another thing, namely, that the radio-humeral joint is quite free. There was absolutely no movement in that two years ago, and the whole thing was a great thick spindle-shaped swelling, and now you will find it is perfectly free from tenderness, and there is no sign of anything wrong whatever. I would like to impress upon you the fact that she has been under treatment two years with splinting and plaster only.

Case 3. *Early Tubercle of Hip-joint.*—Here is a more recent case, namely, a boy who came up here in April on account of the condition of his limb. It was found that he had 20° flexion of his hip-joint, that there was a certain amount of wasting, so that there was a difference of half an inch between the right leg and the left leg, and the same difference between the right thigh and the left thigh. He would not allow any movement in the way of rotation, and there was practically no abduction or adduction. I put him upon a long external splint, and he rapidly began to improve. I will show you the amount of repair that has taken place in two months. This joint was absolutely fixed, and it has been kept still simply, as there was so little wrong in the way of displacement, and I have not even put on an extension. You see the amount of free flexion present. There is the faintest amount of rotation, and all that without pain. The slight fulness which was seen over the front of his hip-joint has gone, the tenderness has gone, and I think he is on the fair way to recovery, though the treatment has been by rest alone. What I shall do with this boy is to keep him at rest for another eighteen months, at any rate, and then let him begin to walk.

Case 4. *Tubercle of Elbow-joint and Humerus. Operation.*—Here is a very interesting little boy. He was handed over to me as a hopeless case, and I was asked to amputate his left arm. He had not only very bad tuberculous disease of his elbow, but he had periostitis and caries running more than half way up his humerus, and he also had two sinuses, so that altogether his arm was in a very bad condition. He was then only eighteen months old. As he had very acute and rapid disease I thought it wise to operate at once. I decided to attempt to save the limb first of all, so I made a long incision down the back of the humerus and elbow-joint, and performed practically a partial excision. I found there was a large amount of disease of the trochlear surface of the humerus, so that was removed. There was a large carious patch running up the outer side of the humerus. The coronoid process of the ulna was practically destroyed, and there was much disease in the upper end of the radius. The whole of the diseased bone was removed and as much of the synovial membrane as possible. It healed wonderfully well, although he was rather a thin, miserable boy. You see that there is now a little sinus in the position of one of the scars. It does not lead down into the joint cavity; it is simply a little tuberculous focus in the skin. He has got a fairly useful joint. It can be flexed up beyond a

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right angle, and almost fully extended, and he has got perfectly free and satisfactory pronation and supination. The operation was performed August 22nd, 1900, and just recently, *i.e.* practically two years later, I let him go without his plaster.

I propose now to say something with regard to the *treatment* of tuberculous joint disease, as illustrated by the cases I have shown you. At the outset one can say that the two great principles that govern the treatment of these cases are (1) *rest*, and (2) failing rest alone, the eradication of the disease by operation. I shall consider these two principles separately.

First of all, as regards treatment by rest, and the question of its efficiency. There is, I know, still a certain amount of difference of opinion as regards what can be done by rest alone. But anyone who is attached to an out-patient department like this for any length of time becomes rapidly convinced that a great deal can be done; and the thing which strikes one very soon is that nearly all these cases of tuberculous disease of joints have a natural tendency to get well, and it is only the result of patients getting about and using the limbs that the disease, apparently, is able to go on at all. As soon as one puts the parts at rest by means of splints, of which I shall have to speak later, the great majority of these patients begin to improve. Apart from hospital patients I do not think there is any doubt that nearly every case of tuberculous disease of a joint will be cured by rest alone under good conditions. And even in bad conditions in the children who are brought here, those who are living in London and are not at all well looked after, the results are so good that I assure you it is comparatively rarely that it is necessary to have to undertake an operation. I have shown you two operation cases and two non-operation cases, but the cases were taken haphazard, and do not in the least represent the correct proportion in practice.

I divide the cases into two stages apropos of rest, namely, into the early stage and into the late stage. With regard to *early cases*, I do not think that there is any doubt, except in acute tuberculous disease commencing in the bones, that the cases get well. I am convinced that considerably over 90 per cent. get well by rest alone. With regard to the later stages of the disease, where considerable destruction has taken place in the joint and about the bones and ligaments, and where often a sinus is present, there are other points to take into consideration. The question of rest here must depend to a certain extent upon the joint which is involved. With regard of that point I should like to mention particularly one or two joints. Perhaps the commonest joint to be affected is the hip, and it is very striking how the hip will get well under suitable rest treatment, even though it is very bad indeed. And if you can possibly get a hip well under those circumstances with rest alone, I am equally convinced that the result as compared with excision is a satisfactory one. Just for the moment let us compare a limb years after excision with one years after treatment by rest alone. I think the chief points are these—that after excision there is nearly always more shortening; one epiphysis is removed entirely, though it should be remembered that the growth of the femur takes place, after the first three or four years, almost entirely from the lower end. After excision of the hip-joint there is usually more movement than after treatment by rest, but, on the other hand, the joint is liable to be rather flail-like, and the limb not so useful to the patient. Therefore, even in the later stages of hip disease, I should prefer to go on with rest; and it is striking that even in very bad cases, after prolonged periods of rest and extension, complete cure may take place. Here I think I might mention an instance which is certainly an extreme one. It illustrates the fact that it is reasonable to hope for success in such cases. Some years ago I was down at the Margate Sea Bathing Hospital, staying there with the resident medical officer, who was a friend of mine. He showed me a hip case. The boy was about twelve years of age, and had had hip disease since he was five or six. His hip was flexed to a right angle, was markedly adducted, the remains of the head of the bone were out of the acetabulum, so there was a pathological dislocation. He had five or six sinuses at different parts of the buttock and thigh. He was much wasted, the liver was enlarged nearly down to the umbilicus, and he had an up-and-down temperature and some albuminuria. He was sent down to Margate as a last resource. My friend said: What sort of prognosis will you give in such a case as that? I said I thought it was hopelessly bad. A year afterwards I saw that boy again, and, simply as a result of scraping out the sinuses, the application of exten-

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sion, sea-water baths, and residence at Margate, his hip had become almost straight, his sinuses were closed, his albuminuria had cleared up, the liver was much smaller, and he had gained greatly in weight. When that can happen in such a very extreme case as this, I think it is reasonable to hope to obtain a good result in milder cases.

Now let me mention another joint in particular, and that is the knee. There, again, it is reasonable to hope that many cases of tuberculous disease will get well without operation. But there is one important exception to this rule. Sometimes in advanced cases of disease of the knee, where no treatment has been carried out, or where unfortunately the wrong treatment has been carried out, as I shall mention shortly, there is a bony displacement. That displacement is usually backward, and also somewhat outwards, so that the tibia is in a flexed position and rests on the posterior aspect of the condyles of the femur. Whatever you do in such a case, nothing short of operation can produce a straight limb. The disease may be cured, and the result may be fairly satisfactory as far as that goes, but the limb cannot be got straight. The reason why this displacement has taken place is that destruction of the posterior crucial ligament has occurred. This ligament, as you know, passes from behind the spine of the tibia in a direction upwards, forwards, and inwards to the internal condyle of the femur, and forms a very important connection between the two bones. If in a dissected knee-joint you try to displace the tibia back on the femur, you cannot do it as long as the posterior crucial ligament is intact. On the other hand, destruction of the posterior crucial is followed by this irremediable displacement owing to the constant pull of the hamstrings. If, then, you see a case in which there is definite displacement of the tibia on the femur, it is necessary to treat it by operation.

There is another special condition which I should like to mention, namely, the presence of sinuses, because then a totally different state of affairs is present. When sinuses have formed, the disease is no longer due only to tubercle, but there is the mixed infection of pathogenic organisms as well, and the amount of destruction which takes place is in excess of that produced by tubercle only, and is very much more resistant to treatment. It is necessary in the majority of such cases to do something more than simply put the part at rest.

The next point that I should like to impress upon you is the great importance of *prolonged* treatment. Often you will find, on treating a patient with tuberculous disease of the elbow, or knee, or hip, for two or three months by means of plaster of Paris or splints, that there will be very rapid improvement; in fact, apparently cure has taken place. But if you leave off the treatment at that stage, what happens in nineteen out of twenty cases is this—that for two or three months perhaps the child will keep perfectly well, and then relapse, either sudden or gradual, takes place. It is therefore of great importance to continue the treatment. If I were asked to mention a time I should say, to be on the safe side, *always keep these cases at rest by one means or another for eighteen months at least after all signs of disease have disappeared.* In the same way, after doing an operation, I have always made a practice of keeping the joint in plaster of Paris at least eighteen months after the operation wound has healed. Many people think that if you keep a joint at rest sufficiently long it becomes stiff, but that is not so. As a matter of fact, the longer you keep these tubercular cases at rest, the better. In the elbow case that I showed you, the elbow was stiff at first, but now, after eighteen months' treatment by plaster of Paris, there is comparatively free movement in the joint.

There is a hip-joint case which should have come up this afternoon, but he has not done so. It is most interesting. He has had a number of operations done on his hip, and luckily it was treated by prolonged rest afterwards; rest and extension were applied for two years after the last operation. That boy has a joint which is perfectly sound, and which has more than half the normal degree of mobility.

In order to further impress upon you the value of rest, I made a few notes of a very interesting paper which was written by three New York surgeons, giving an analysis of 150 cases of hip-joint disease which were treated at the New York Hospital for the Ruptured and Crippled. This analysis was made something like twenty years after the first case was treated, so the cases were spread over a long period. Many had been cured for many years before they were reported in this paper. Twenty-five of those 150 cases were still under treatment

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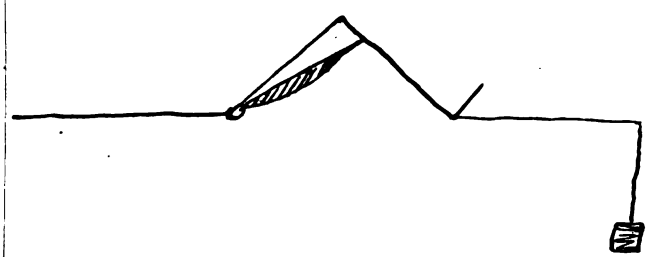
when the paper was written, so we have to consider the result in only 125 cases. The treatment consisted in rest and extension; when abscesses formed they were either aspirated and injected, or they were opened, curetted, and sewn up again. In only four cases was excision performed. So we can practically leave excision out of the question. As regards final results in these 125 cases, eleven of them died and 114 were cured. One hundred and seven out of the 114 cases had strong useful limbs and a greater or less degree of mobility, and were able to follow active employments. The other seven were put in a separate group because they had a considerable amount of deformity. They were able to earn their living, but they were considered to have such deformity that they were advised to enter the hospital and have the deformity treated. With regard to these cases, again, taking shortening as an index, it struck me that these results were extremely good, compared with any group of excision cases. In twenty-one cases there was no shortening at all. In seventy-one it was under an inch, and in thirty-six it was over an inch and under one and three-quarter inches. I also made a note about the amount of deformity as an index of the efficacy of the treatment. Forty-seven cases had no deformity at all, seventy-seven had under 10° of flexion, an amount which you can practically neglect, and in thirty cases there was 10° to 30°. I am certain no surgeon could expect better results than these, and after excision I do not believe that the results could be nearly so good.

I will now say a word or two with regard to the manner of applying this principle of *rest*. In the majority of joints it is quite sufficient to keep the joint at rest by means of a splint or, preferably, plaster of Paris, which, in my opinion, is better than anything. On the other hand, in the case of two joints, it is necessary to do something more.

The joints I refer to are the hip and the knee. The hip, as a result of muscular spasm, becomes either flexed and abducted or flexed and adducted, and the knee tends to become strongly flexed. In those two instances it would be wrong to allow the joint to settle down in that position, because they are such extremely awkward positions with regard to subsequent use. So something special must be done in the case of the hip and knee, and of course the special treatment I refer to is *extension*. The objects of extension are, first of all to keep the parts at rest; secondly, to prevent pressure taking place between the bony surfaces; and lastly, to allay muscular spasm, and so overcome deformity.

As regards methods of application, most of you know perfectly well how to apply a stirrup, and put the extension on to the knee or hip; but the extension is so often applied wrongly that I do not think I shall waste time if I illustrate to you the fact that there is a right way and a wrong way of applying it. Let us take the knee first. Supposing you have a knee which is flexed. Extension is sometimes applied here as shown in this diagram (Fig. 1), in the hope that it will gradually overcome the spasm of the muscles which is causing the deformity, and produce a straight knee. But think for a moment what takes place. The muscles producing the deformity are the hamstrings; they

Fig 1.



pass from the back of the pelvis to the upper part of the tibia (*vide* Fig. 1). When extension is made you convert the leg into a lever, the attachment of the muscle to the tibia being the fulcrum. The effect is to press the articular surface of the tibia against the condyles of the femur, and so produce interosseous pressure, which sets up more pain and increases the muscular spasm. Further, the upper end of the tibia is forced in a backward direction, and so a strain is put on the posterior crucial ligament, which, if softened by disease, may give way, and a dislocation result.

That is the wrong method to which I referred when I spoke of dislocation the result of disease. Now, what is the correct way to treat a knee which is deformed in that way? The correct plan is to apply extension as shown in this diagram (Fig. 2). The effect of the extension is then directly on the knee-joint itself, and it tends to separate the two bones and at the same time to tire out the muscles

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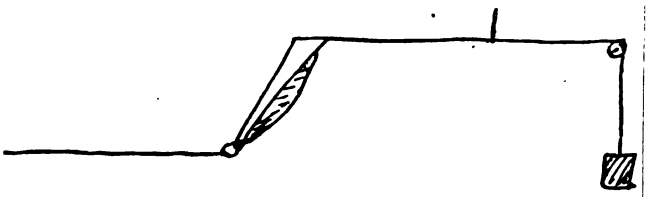
that are producing the flexion. If you leave it in this position for a week you will probably find that you can straighten it 20°. It is then brought down to this extent, and the process continued until the limb is straight.

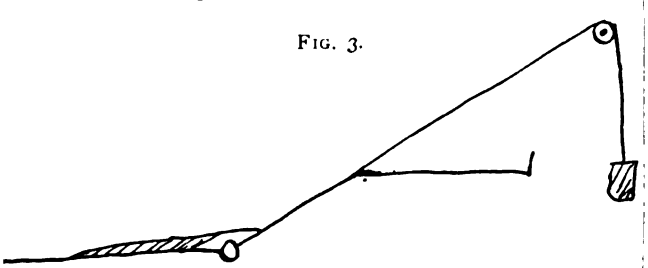
The same thing applies to the hip. Here, however, it is even more common for the extension to

FIG. 2.



be applied wrongly, so that interosseous pressure is produced, and hence the pain and spasm increased. The correct plan is again to apply the extension in the line of the limb in its deformed position. For instance, if flexion alone is present, extension should be applied as shown in Fig. 3, the leg being supported upon a firm pillow or frame of the proper shape. In this position you will see that the psoas muscle will no longer act as a fixed point, but the effect of the weight extension will tend to tire out the psoas and separate the bones, namely, the head of the femur, from the acetabulum. After a very few days you will find that you can diminish the flexion, and so gradually get the limb straight. Abduction or adduction must, of course, be dealt with on corresponding lines. At the present time I have a boy in Guy's Hospital who had been allowed to get about as best he could with bad hip disease. He came to me with a hip

FIG. 3.



which was very strongly abducted and very markedly flexed. He has had extension on for five weeks, and at last we have got it straight. All his pain has gone, and movement is free.

Now we come to the question of operative treatment. If rest does not suffice it may be necessary to do something in the way of operation. The first question which always arises is: How is one to decide that it is no longer any use continuing the rest, and that it is necessary to perform an operation? The following answer to the question is, I think, a fair one: If you put the patient under the best possible conditions and fix the joint, and if necessary apply extension, and if at the end of a month the symptoms and physical condition of the joint are distinctly *worse* than they were at the start, it is absolutely necessary to do something further. I do not think that it is of much use to try rest and fixation for longer than a month if the disease distinctly progresses in spite of it. If you can send your patient to Margate and try another month there, the chances are that you will get him well; but one cannot send all or nearly all one's patients down there, so we have to do the best we can for them. Now, the question arises as to what operation should be performed.

I shall not now enter into this question in detail, but I will content myself by indicating to you the main principle that I consider should guide one in such operations. The aim should be to as far as possible remove all the diseased tissues with the least possible amount of interference with healthy structures. From this you will gather that I am in favour of what is sometimes termed erasion wherever possible, rather than a set operation, such as a formal excision of a joint, in which a large amount of healthy tissue is frequently sacrificed. Finally, I would impress upon you the great importance of *prolonged rest* after whatever operation is performed.

You have been able to see for yourselves in the two operation cases that I have shown you that the kind of operation that I have referred to will, if carried out thoroughly, give satisfactory results even in very severe cases.

ONE of the most troublesome and lingering symptoms in the slighter attacks of influenza is a hard, distressing laryngo-tracheal cough, with scanty, tenacious expectoration. Ordinary sedatives often aggravate it, but this mixture has been used with success:—℞ Apomorphinæ Hydrochloridi, gr. j; Morphinæ Hydrochloridi, gr. ¾; Acidi Hydrochlorici Diluti, ʒss; Syrupi, ʒiv; Aquæ ad ʒ iv: M. f. mist. One or two teaspoonfuls for a dose. (Burney Yeo.)

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A CLINICAL LECTURE
ON
ABDOMINAL TUMOURS.

By E. STANMORE BISHOP, F.R.C.S.Eng.,
Hon. Surgeon, Ancoats Hospital, Manchester.

GENTLEMEN,—Someone has likened the abdomen to South Africa—both, he says, are full of surprises, and this, at least as far as concerns the abdomen, is perfectly true. Abdominal surgery has thrown much light in the past few years upon many of the dark places in it, and certain rules upon which we may with fair safety depend are becoming more clearly definite. A tumour of the abdomen can now be pretty certainly located, and once this has been done, it becomes a much more easy matter to determine what at least it will probably prove to be. The possibility of surprises, however, is not yet eliminated, and therefore there is still considerable interest attaching to every abdominal section.

It will be well first to specify some of the rules upon which we depend for approximate diagnosis in certain cases, for definite assurance in others.

An abdominal swelling is either situated in the abdominal wall itself or in the cavity contained within. If a patient in the dorsal position makes an attempt to rise into the sitting posture without the aid of his hands, he must necessarily contract the muscular layer of his abdominal wall. As all growths or swellings in this wall lie in or above this layer, they must with equal certainty be forced forward and become more prominent by such contraction. On the other hand, as such contraction renders the wall more resistant, any swelling lying beneath will become less prominent, less apparent to the examiner's hand placed upon it. In this way we have a clear diagnostic sign separating these two classes of swellings.

Of swellings situated in the abdominal wall itself, hernias form by far the largest class. As the openings through which they pass occupy certain well-known anatomical positions, there is seldom much difficulty in keeping them distinct from all other swellings. The only exceptions to this are ventral hernia, lumbar, and vaginal or pudendal hernia. Ventral hernia is almost unknown, except as a sequel to some abdominal operation. The presence of a scar over or near it will therefore suggest its nature.

Lumbar hernia is rare. When it occurs, it is found just above the iliac crest which forms the lower wall of its triangular canal—the canal of Petit. The other two walls, which incline towards one another above, are the internal oblique muscle in front, and the latissimus dorsi behind. Its position is about the junction of the posterior with the middle third of the iliac crest.

Pudendal hernia, of course, is only found in women. It also is rare. It projects the mucous membrane lining the vagina before it, lies on one or other side, and passes outwards beneath the pubes, bulging, if large, one of the labia majora.

All hernias may be distinguished from other mural swellings by one fact. Being temporary displacements of organs, originally formed in the abdominal cavity, there must be an organic connection between the protrusion and the parts beneath. No posterior definite line of demarcation can therefore be felt. The fingers of the examiner, however deeply placed, can never get actually behind it. This is not the case with any other swelling occurring in the abdominal wall.

Hernias usually contain intestine or omentum, but they may contain almost any movable abdominal structure. They are, therefore, whilst usually resonant on percussion, not invariably so. If reducible, the very fact that they can be made by pressure to disappear confirms the diagnosis. If not reducible, and containing some solid body, as an ovary, or undescended testicle, there may be room for hesitation. Sometimes, in such a case, operation alone can decide the question. Secondary points then become important. The absence of a testicle on the affected side, increased swellings at and before menstruation, and the fact that the posterior extremity of the solid body cannot be felt or outlined by the fingers, will throw additional light upon the question. Other mural swellings are either composed of overgrowths of the tissues normally present, as fibromata, lipomata, neuromata, or myomata, or are malignant or syphilitic.

But the main interest naturally centres upon swellings which lie *internal* to the abdominal wall.

One of the first points to determine of any swelling which is manifestly by the above test not

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in the abdominal wall is whether it is common to the entire peritoneal cavity, or enclosed in and walled off by a distinct boundary from that cavity. If common, is the distension due to fluid, or to air? If to both, diminution of the normal liver dulness will point to perforation of the digestive tract, at some point, which will probably be determined by a careful consideration of the history of the case.

If the swelling is clearly marked off from the general cavity by a distinct boundary line, we are justified in assuming that it is produced by a tumour. The next step is to determine the character of its contents, whether fluid or solid. In settling this point, puncture is never justifiable. Paracentesis abdominis is every year less and less required. Only in ascites is it permissible, and even here, the advantages to be obtained by laparatomy combined with union of the omentum to the abdominal wall, in preventing refilling and ameliorating the results of backward pressure in the portal vein, render this blinder method of relief less advisable. But in any case where the contents of an abdominal swelling are plainly encysted, puncture is not only obsolete; it is almost criminal. The dangers of the practice cannot be too strongly insisted upon. The character—fluid or solid—can usually be easily determined by the trained hand. Palpation for this purpose should be gentle and most patient, the tumour itself being either supported by the examiner's other hand or pressed against some resistant portion, as the spine, of the patient's own body.

Much may be learnt by considering the mobility or otherwise of the tumour. If mobile, is it affected by the descent of the diaphragm in forced inspiration or not?

Those which move readily with respiration must be in close connection with the diaphragm. Thus hepatic and gall-bladder swellings move most of all. Not only do they move downwards with deep inspiration, but they move upwards again—and cannot be prevented from so doing by the examiner's hands—with expiration. The same is true of gastric swellings when they involve the upper part and body of the stomach, but not when they involve the pylorus. Splenic and renal swellings, not being so intimately connected with the diaphragm, move downwards with forced nspiration, but move upwards again somewhat sluggishly, or not at all, and can often be held by the examiner's hand in the position which inspiration has given them.

Then, of swellings which do not move synchronously with respiratory movements, we have those which are palpable and visible on inspection, and those which require bimanual examination in order for their detection.

The former may be immovable, or, on the contrary, they may move freely in any direction. Even the action of gravity may apparently alter their position greatly. Such tumours are usually seated in the omentum, small intestine, or mesentery. If the patient is placed in the upright position, the tumour, which was centrally situated when its owner was lying flat on his back, will now be found in the hypogastrium, or in one or other iliac regions; whilst if the foot of the bed be lifted sufficiently, or, still more, if the patient be placed in the Trendelenburg position, the swelling will be found to have migrated to a new location beneath the ribs on either side. Such complete mobility can only be found in a sub-peritoneal fibroma or ovarian growth, the pedicle of which has yielded, or in an omental, mesenteric, or intestinal growth.

Without any of this exaggerated mobility, tumours may still be capable of movement, either by the examiner's hand or by changes in the position of the body. Inasmuch as any mobile tumour must have, if only for the entrance of blood-vessels, some connection with a more fixed part, information as to its point of origin, which will be that of this blood-supply, is forthcoming if the area through which this movement takes place is carefully noted. A given point on the surface of the tumour furthest from the point of blood-supply must during its movement describe an arc, which will be part of a circle, the centre of which must lie at the spot at which these vessels enter. This spot can thus be determined, and the organ which normally occupied such a location will almost certainly be found to be that from which the growth has arisen. The organ once identified, the kinds of growth peculiar to it may be passed in review, and their differentiation effected by other observations to be presently referred to.

Swellings of the abdomen may not only move with respiration, or be capable of movement by the examiner's hand; they may possess independent movement. This is, of course, most marked in

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those connected with the digestive tract; and this movement may be very slow, or sufficiently fast to at once attract attention. In acute obstruction the small intestine becomes dilated, and distends the abdominal wall. Peristaltic wave-like movements may be easily watched, causing the abdominal wall itself to rise gradually and fall again as if a ripple were passing over its surface. In chronic obstruction this is still more marked, as sufficient time has been afforded the gut above the block to become hypertrophied. Moreover this form of obstruction usually affects the large intestine, and for both these reasons the result is more prominently manifested. In acute intussusception, during a paroxysm of pain a tumour may apparently develop and, as the pain becomes less, fade away and almost disappear. In chronic intussusception, whilst no appreciable alteration in place occurs during any one observation, the tumour will be found to have materially altered its site if successive observations are carefully recorded for a few days. Fæcal masses supply perhaps the most startling changes of site, however. Apparently at the first examination they may be as hard and fixed as carcinoma, and the next day have either entirely disappeared or occupy a perfectly new situation. This was well exemplified in a case seen lately. A lady had symptoms of pyloric obstruction, and both medical attendant and consulting physician satisfied themselves not only of the presence of a hard nodular mass at the pylorus, but of another, apparently affecting the body of the stomach, lying more in the left hypochondrium. Whilst it might have been possible to resect the pylorus, the presence of the second mass appeared to negative operation; and a surgeon was called in two days later to decide this question. The second mass had then entirely disappeared.

In the opposite direction, perfect immobility almost always points to a growth arising behind the peritoneum. It is a universal law that all intra-peritoneal structures are capable of movement to a certain extent, and therefore tumours arising from them, at least in their earlier stages, partake of such mobility. When a growth, therefore, is absolutely fixed, its point of origin is almost certainly behind the peritoneum. Of course, in speaking of post-peritoneal or intra-peritoneal structures, one is speaking clinically. From a purely anatomical or embryological standpoint, every structure is post-peritoneal. Such immovable post-peritoneal tumours, if not aneurismal or pancreatic, are sarcomatous or syphilitic. Renal tumours, although post-peritoneal, are usually movable to some extent. Retro-peritoneal glands, though they may be enlarged, are seldom sufficiently so to be detectable by palpation, still less by inspection without laparotomy; and although a calculus in the mid-course of the ureter behind might theoretically be considered capable of being palpated, I have never been able to do this until it has reached the pelvis, nor am I aware that it has ever been certainly done.

The outline of the abdomen viewed from the side—the examiner's eye being on the same level—the sky-line, so to speak, will often give information. Here, too, the movements of respiration are to be studied. In a perfectly normal abdomen the sky-line moves synchronously with inspiration and expiration, rising with the former, sinking with the latter. Should no tumour be present, the movement is seen all along the line from xiphoid cartilage to pubes. Like a violin string, vibration is communicated along its whole length, but if a tumour is present beneath sufficiently large to press against the abdominal wall, vibration ceases at this point, just as a fiddle string ceases to vibrate at the bridge or at any point stopped by the pressure of a finger.

Certain swellings have almost characteristic sky-line outlines. Thus the swelling produced by ascites, if the abdomen is not fully distended, is flattened in the centre, whilst bulging in the renal regions. If distension is complete, the whole sky-line is convex equally from xiphoid cartilage to pubes; the abdomen projects as a whole. The swelling of a pregnant uterus also projects prominently forward in the centre, but the sides are flattened, and the sky-line is flattened near the xiphoid, bulging out firmly an inch or two below.

The manner in which the sky-line ends is especially to be noted. In pregnancy this line rises gradually from the pubes, but dips down abruptly near the xiphoid. In fibroid disease of the uterus the dip is as abrupt towards the pubic as towards the xiphoid extremity. There are two main forms in this disease, both with the marked abrupt rise from the pubes; in one the outline above this rise is a regular curve, as if over a large round ball; in the other, irregular, as if covering

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several differently sized mounds. The rounded, even form may be distinguished from ovarian tumours by the fact that whilst in both the rise from the pubes is abrupt, that from the xiphoid end is abrupt also in fibroids, but gradual in ovarian cysts.

Enteroptosis is easily distinguished from either of these, especially in the upright position. In all other conditions the abdomen projects abruptly forwards, the hypogastric region being always above the upper line of the pubes. In enteroptosis it falls forwards and downwards, and the hypogastrium lies below the line referred to. Moreover in enteroptosis there is what is called a "bridle line," a deep wrinkle in the abdominal wall, commencing above either iliac crest in the flanks, where it is but slight, but deepening and becoming much more marked as it passes downwards to its lowest level, about one inch above the pubes in the centre. In no other condition is this seen, and its intensity pretty accurately marks the extent to which the abdominal contents have descended. Enteroptosis may, of course, coexist with intra-abdominal tumours, but the tension produced by them rather tends to obscure the sign of enteroptosis than *vice versâ*.

Tumours which require bimanual examination for their detection are so many and various that even the most general description of them and their differentiation will require a separate lecture.

E. FRAENKEL describes the case of a four-year-old boy, dead of scarlet fever, whose entire œsophagus and stomach, excepting only a small area under the cardia, were in a state of necrosis and ulceration, resembling the condition which follows ingestion of a caustic. Another case of marked ulceration involving the lower half of the œsophagus occurred after scarlet fever in a girl eighteen months old, and an earlier stage of the process was observed in a girl of five. Microscopical examination showed that the process was one of necrosis without suppuration, and streptococci in large numbers were demonstrated in the diseased areas. The process is to be regarded as a secondary one, due to the abnormal condition of the tissues in scarlet-fever patients. The streptococci undoubtedly came from the surface of the gastric mucous membrane.—*Amer. Journ. Obstet.*, February, 1903.

CLINICAL LECTURES

ON

DISEASES OF CHILDREN.

LECTURE VI.

TUBERCULOSIS IN CHILDHOOD.

Delivered at the London Hospital, November 4th, 1902.

By ROBERT HUTCHISON, M.D.Ed., M.R.C.P.,
Assistant Physician to the Hospital, and to the Hospital for Sick Children, Great Ormond Street, W.C.

GENTLEMEN,—In the last lecture we were considering wasting as a sign of disease in infancy, and we saw that there were several morbid conditions which may be associated with it as one of the chief symptoms. I enumerated to you several possible causes of wasting in children which you must consider when a case comes before you, and I dwelt particularly upon congenital syphilis as one of the organic diseases which may be a cause —sometimes an unsuspected one—of that condition. Now in this lecture I wish to direct your attention to the other great organic disease which may be a cause of wasting in children, both in infancy and in later childhood; I mean tuberculosis.

The frequency of this disease in children will be realised from this fact: that careful hospital statistics, taken in various parts of the world, show that about one third of all the children who die in hospital die of tuberculosis in some form or another; and further, in about twelve per cent. of the remaining cases tubercle is present in a latent form. If you examine the bodies of those children after death, you will find that the tuberculosis tends to attack one particular set of tissues with a very striking degree of regularity. Those are the tissues which are concerned with the formation of the blood. Ninety per cent. of all the cases show an affection of the lymphatic system or the bones. The lymphatic system, you will remember, is engaged in the formation of one variety of white blood-corpuscle, and the bones are quite as much blood-forming organs as they are supporting tissues. And so when one says that the bones are affected, one really means that a blood-forming organ is affected. And I want to impress upon you that fact: that it is this particular set

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of tissues which, more than any others, tend to be involved in tuberculosis in early life. If you look at such a diagram as this, which shows the age distribution of 500 consecutive cases of tuberculosis in children, taken from the statistics of Great Ormond Street Children's Hospital by Dr. Still, you will see that tubercle attains its maximum frequency in the second year of life. And I want you to correlate in your minds those two facts; firstly, that it is the blood-forming tissues that are specially affected by tubercle; and secondly, that it is in the second year of life that tubercle attains its maximum frequency; because I think there is a relationship between them which is not an accidental one. I think it is probably owing to the blood-forming organs being in a very active state of vitality at this period that explains their liability to tuberculosis. When I come to speak of the anæmias of infant life I shall have occasion to point out that the blood of children undergoes a very important transformation in character at just about this time, towards the end of the second year. It is about that time that the tissues which form the white blood-corpuscles come to attain their greatest degree of activity. After that the lymph-forming organs gradually pass into the condition in which they are found in the adult.

Of more importance for us, however, is it to study how the tubercle bacillus most commonly finds access to the body in childhood. It is all very well to know that the tissues most involved are the lymphatic or blood-forming organs; but what we specially want to know is how the bacillus gets there at all. Of a series of 216 cases which were carefully studied from this point of view by Dr. Still, it was found that the access of the bacillus had been through the lungs in 63·8 per cent., through the intestine in 29·1 per cent., and through the middle ear in about fifteen cases. These statistics were derived from the Great Ormond Street Children's Hospital, but there are similar statistics from other children's hospitals. They all tend to show the same thing, namely, that the lung is by far the commonest path of infection. You will see at once what the significance of that is. It means that the talk about the milk of tuberculous cows being the chief cause of the infection of children with tubercle is certainly wrong, and it also means that dust must be regarded as the main source of infection in the child, just as it is in grown-up people. The tubercle bacilli are conveyed into the lungs, probably mixed up with dust, and thence they find their way into the lymphatics. When one speaks of the lung in this connection it must be remembered that the chief part affected is the bronchial glands at the root of the lung. But these are not likely to be affected if they are perfectly healthy. Unless there be a great inherited predisposition to tubercle the healthy gland may be assumed to be able to deal with such tubercle bacilli as find access to it; otherwise all of us would be suffering from tuberculosis. And the reason why the bronchial glands in children become so frequently affected no doubt is that they are very often not in a healthy condition; they have already been the seat of inflammation. How do they become inflamed? They become inflamed secondarily to catarrh of the bronchi or alveoli. In other words, the bronchial glands become inflamed during an attack of bronchitis or broncho-pneumonia. The bronchitis or broncho-pneumonia is recovered from, but the glands are left in an unhealthy condition, dust containing tuberculous material is inhaled, and the bacilli start to grow in the glands and set up a caseous degeneration of them, which is the starting point of tuberculosis. So I want to impress upon you as the great predisposing cause of tubercle in children that group of diseases which are accompanied by catarrhal processes in the bronchi and lungs, particularly such diseases as measles and whooping-cough. And you will constantly find in your practice that children will be brought to you for wasting and some vague complaint of ill health. You inquire into the past health and find that they have had an attack of measles some months or weeks before, and have not been well since. Then, when you come to make a physical examination, you will find that they are the subjects of tuberculosis. That is a very common history.

Now no matter by which path the tubercle finds access, no matter whether it is through the bronchial glands or through the intestines, affecting the mesenteric glands, or through the middle ear, it sooner or later generalises itself. And that is the other point I want specially to emphasise,—that no matter what the primary path may be by which the child becomes infected, the disease has a great tendency to become wide-spread, a much greater

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tendency than is the case in grown-up persons. What usually happens is that one of these caseous bronchial glands softens; it ulcerates its way into a bronchus, and the caseous material gets aspirated into the lungs, and so pulmonary tuberculosis is set up. In other cases perhaps the infection gets conveyed by ulceration into a blood-vessel, and is disseminated in that way through the body. At all events, in whatever way dissemination occurs it takes place very quickly. That is why it is so important for you as practitioners to diagnose tubercle in children as early as possible; because, unless you recognise it early and treat it promptly, it will certainly before long become generalised, and then it may be too late to treat it at all.

That leads us naturally to consider how you are to diagnose the existence of tuberculosis in children. I shall consider first of all the diagnosis of *general tuberculosis*. We will assume that the process has already become generalised. How are you to know, then, that the wasting in this particular case is due to general tuberculosis or not? Often, I believe, it is not possible for you to know at all. It is not possible to know until the post-mortem in very many cases. The diagnosis of general tuberculosis in children is extremely difficult, and it is so for this reason: that the process kills the child before the tubercles have had a chance of breaking down. You know that unbroken down tubercles do not give rise to physical signs. You may have a lung stuffed with miliary tubercles, but until they soften and break down you have no means of telling that they are there, and it is on this account chiefly that the diagnosis is often so uncertain. There are, however, certain symptoms and signs which should make you suspicious. One of these suspicious symptoms is the occurrence of irregular pyrexia along with wasting, for which you can find no other cause; again, if there is much apparent illness and fretfulness on the part of the child, without obvious or apparent reason, you should be suspicious that it is a case of tubercle. But there is no other symptom to help you much. You will easily see that the danger you run is of attributing those symptoms to, perhaps, a slight dyspepsia, or catarrh of the stomach or intestine; or, perhaps, to a slight bronchial catarrh, or to some trivial condition of that sort which may be present along with the tuberculosis, and not to tuberculosis itself. It is so extremely difficult to assign its proper value to each of these symptoms that you have often to suspend your judgment.

Amongst the *signs* which may give you some assistance in the diagnosis of tuberculosis are certain appearances in the child, appearances which clinical experience has shown to be often associated with tubercle. Tuberculosis is known to be commoner in children of a certain type. You know the fragile, fairy-like type which is supposed to be particularly marked out for tuberculosis. That is an extreme example of what I mean. In young babies, however, that type is not so noticeable, and you have to go by such minor indications as long eyelashes, or the presence of an excess of downy hair, especially on the back between the scapulæ. There is also, which you will find of value, a tendency to the appearance of blotches of erythema upon the skin on very slight pressure or irritation. These signs may perhaps seem to you trivial, but very often, taken together and along with the family history and the general symptoms, they may suffice to put you on the right track. Of course you will look also for signs indicating the starting point of the process. First of all you will look for signs of the disease having started in the lungs. I shall describe to you immediately what the signs associated with tubercle of the bronchial glands are; but I shall only say here, that in this case again you have great difficulty in coming to a right judgment, because, admitted that you get moist sounds over the lungs in a case which you suspect may be one of tubercle, you are still in doubt as to whether that is due to breaking down of tubercles or not, because there is nothing commoner in marasmic children than for them to suffer from a slight attack of hypostatic pneumonia as they lie in bed, and that may easily give you moist sounds over the back. So it is only when you get signs of breaking down on the anterior aspects of the lungs that you should attach much importance to them.

I next pass to the consideration of the physical signs associated with *tuberculosis of the bronchial glands;* in other words, the signs which you find when the tubercle finds access, as it usually does, by means of the lungs, and when it has not yet become generalised. Here, again, there is no quite conclusive symptom. There will be more or less wasting, and perhaps there will be irregular pyrexia. There will also be general fretfulness

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and illness. But the only local symptom of any value is the occurrence of a spasmodic cough. Children with enlarged bronchial glands often suffer from such a cough, which you will be apt to mistake for whooping-cough, which it may simulate very closely. Many cases of whooping-cough which are supposed to drag on for months are probably cases which may have begun in whooping-cough, but which are now suffering from secondary enlargement of bronchial glands. Note, then, that one point which will help you is the existence of this spasmodic cough. When you come to the physical signs you will find that they are only present in a minority of the cases, and you have no difficulty in seeing why that should be. The bronchial glands are not very large; they lie deep down in the mediastinum, and it requires a great deal of enlargement of them to produce signs which you can recognise with certainty. So you must be prepared to find that in cases in which after death you are able to recognise a considerable degree of enlargement of the bronchial glands, there were no signs of them during life. Still, in some cases, when the enlargement is very great it will reach the surface, and the chief sign in such a case is dulness behind the manubrium sterni, dulness which extends outwards towards the apices of the lungs, and downwards towards the heart. I would warn you, however, of one possible fallacy here—namely, that enlargement of the thymus in children may exactly simulate this sign. There is no way of discriminating exactly between the two. Another sign which you will find is distension of the veins in the neck. This is due to the fact that the enlarged bronchial glands tend to press upon the superior vena cava. Further, owing also to such pressure upon the veins, there may be a bruit heard on listening over the manubrium. Dr. Eustace Smith has pointed out that this is best heard if the child stretches its head back, which causes the superior vena cava to be pressed upon by the mass of glands. But I do not think you should attach too much importance to that sign. One certainly hears a bruit of that sort in many cases in which there is no reason to suppose that the bronchial glands are enlarged. On the other hand, its absence is an indication of some value, which should make you suspicious that you are not dealing with such enlargement. I am afraid these are the only signs which one can give you as indicative of enlargement of the bronchial glands, and you will see that, taken altogether, they are not of very great value. What I wish to impress upon you by all this is, the great difficulty which you will have in making up your mind about either bronchial tubercle or general tuberculosis; and you must often remain in doubt, and must not blame yourselves too readily if, after death, you find extensive tuberculosis when you have been regarding it as a case of ordinary wasting.

The next form of the disease which we come to is much more hopeful, both as to diagnosis and treatment, namely, tubercle in the abdomen—those cases in which the bacillus has gained access through the alimentary canal into the intestinal glands and peritoneum. Abdominal tubercle differs so much from tubercle elsewhere in children—and the same is true of grown-up people—that one is tempted to suppose it is a different disease; and it has recently been suggested that it may prove to be bovine tuberculosis in man, and therefore a milder form of a similar pathological process. If that were established it would at least explain the very marked clinical differences between abdominal and other forms of tubercle. When we come to consider prognosis and treatment, you will find that abdominal tuberculosis is much more curable than any other form; indeed, one takes quite a cheerful view of it, because a large proportion of cases get perfectly well.

Abdominal tuberculosis resembles the other forms in being most common in the second year of life. Here are the statistics of a number of cases of fatal tuberculous peritonitis in children, collected by Dr. Still when he was registrar of the Great Ormond Street Children's Hospital. You will see that the maximum frequency of death occurs in the second year. That is against the milk theory. Seeing that children live almost exclusively on milk up to the first year of life, one would expect abdominal tuberculosis to be common in the first year if milk were the main source of infection. Not only does abdominal tuberculosis occur most commonly in the second year of life, but at that particular time the abdominal tubercle is usually only part of a general tuberculosis. In young infants it is not purely abdominal tubercle which you have to deal with. You may think when you examine the case that it is abdomi-

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nal tubercle only, but you will almost certainly find that there are tubercular deposits elsewhere. That is only another way of saying that tubercle in young infants, no matter where it starts, tends rapidly to become generalised, and if it starts in the abdomen it ends by involving the lungs or the meninges of the brain; and what you will find constantly happen in these cases is that the case drags on until of a sudden it flares up into tuberculous meningitis, which is followed by death.

There are two varieties of abdominal tuberculosis. There is the plastic form and the ascitic form. Most of you must be quite familiar with those two varieties. The plastic form consists in an enlargement of the glands and a matting together of the coils of intestine with tubercular deposits in the peritoneum and mesentery. The ascitic form is that in which an effusion of serous fluid takes place into the peritoneal sac.

To the first or plastic form the term tabes mesenterica is sometimes applied, but I think quite erroneously. I strongly advise you to discontinue the use of that term altogether. For my own part, I have hardly ever seen a case of true tabes mesenterica. It is a disease which, if it occurs at all, must do so with extreme rarity. Rightly used, the term tabes mesenterica means an enlargement of abdominal glands, with caseation, and nothing else. That may occur, and probably does sometimes occur, but it is very unusual. It will certainly conduce to clearness if we talk merely of the plastic type of abdominal tuberculosis. This type is commoner than the ascitic. Some people say that it is ten times commoner, but my own observation would not corroborate that, I think. In older children at least the ascitic form is certainly commoner than that; it is much more than one tenth of the other. You will recognise this form by the general signs of tubercle, the wasting, pyrexia, and so on. The local signs are hardness, with nodulation, in the abdomen. You may take it as a broad general rule that if you find nodules in the abdomen of a child, if you can exclude fæcal masses, the probabilities are enormously in favour of those nodules being tuberculous, no matter where in the abdomen they are situated, and no matter what they feel like. Tubercle is so very common compared with anything else that you do wisely to regard every mass in a child's abdomen, where you can exclude a fæcal accumulation, as being a tuberculous deposit of some sort. You can easily exclude or confirm a fæcal accumulation by the administration of purgatives and enemata, and if you are dealing with a doubtful case of abdominal tuberculosis you should take that precaution, especially seeing that the large bowel is thoroughly emptied. Then examine the abdomen again, if need be under an anæsthetic, and see if you can still discover the lumps. They are due partly to caseous deposits in the mesentery and elsewhere, partly to glandular enlargement, and partly to coils of bowel bound together by adhesions, and are the main physical sign of the plastic form of abdominal tuberculosis.

The ascitic form you recognise by the presence of fluid. Is the ascitic form of tuberculosis ever simulated by any other peritoneal effusion? In other words, may there be clear fluid in the abdomen of an infant which is not due to tubercle? Of course such effusions may sometimes occur in the course of cirrhosis of the liver, and you may find a difficulty in excluding the presence of that disease. Not long ago I had the case of a little girl in whom that problem came before one in a very acute form. She looked the picture of robust health, but her abdomen was full of fluid, and the liver was about two fingers' breadth down. Its edge was also very hard. It was thought to be a case of cirrhosis, and that it would be well to treat the case from that point of view. However, one watched the patient, and she developed a temperature, and then one began to be suspicious and thought it would be better to operate, because if it were tubercle it would be the right thing to open the abdomen; and, even if it were ascites due to cirrhosis, operation would not do any harm. On the day decided upon for operation she suddenly became comatose, developed all the signs of tuberculous meningitis, and died; and post mortem the abdomen was found to be the seat of very generalised tuberculosis. Such a case may cause you difficulty; but I would again remind you of the great frequency of tuberculosis and the comparative rarity of cirrhosis; so it will be safer when in doubt to diagnose tubercle. Clear effusions into the abdomen due to simple chronic peritonitis have also been described, but they are very rare. They are said to occur after measles or scarlet fever, or after injury. Many good observers, however, are of opinion that all clear effusions of an

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inflammatory sort into the abdomen in children should be regarded as tuberculous.

Both forms of abdominal tubercle are commonly accompanied by ulceration of the intestine; according to some statistics, in as many as 70 per cent. of all the cases. Ulceration does not necessarily show itself by clinical symptoms. It does not necessarily cause diarrhœa. But where ulceration becomes extreme and extensive you will find it tends to be accompanied by an extreme degree of anæmia, a degree which may almost resemble pernicious anæmia in its intensity.

We turn now to the general treatment of tuberculosis; but one may first say a word about prevention, which of course is more important than cure. I need not remind you of the general hygienic rules which should be observed in the rearing of children: attention to proper feeding, proper exercise, proper ventilation of the sleeping rooms, and all those other commonplaces which even lay people now recognise. But I would direct your special attention to the question of dust, because if dust is the chief cause of infection, as statistics show it to be, then dust must be one of the chief things to fight against. And I think there is no doubt that, just as in the last century sanitary reformers fought against a bad water supply and bad drains, so in the early part of this century they will have to make a fight against dust. "Down with dust!" must be the motto of those who would prevent tubercle either in children or grown-up persons. There is here a boundless field for the energies of the reformer. Another point is this: seeing that dust infects through the bronchial glands, what you have to do, if you want to prevent children becoming tuberculous, is to keep their bronchial glands healthy. To do that you have to treat every case of catarrh of the lungs or in the bronchi seriously. You must not think that even a slight bronchitis is a trivial thing, nor yet a little attack of broncho-pneumonia. As far as you can you must see that the child's convalescence is made thorough and complete; that there is not left behind a little wheezing, or a few crepitations at one base. You must try and restore the child to complete health. As it is certainly possible for the abdominal form of tuberculosis to be conveyed by the milk of tuberculous cows, you should, of course—particularly in the case of children who are predisposed by heredity or by surroundings to become infected with tubercle—see that the milk which they get is at all events boiled; I do not say it should be necessarily sterilised, but it should be boiled.

With regard to the treatment of different forms of tubercle, I need hardly say there is no special treatment for the generalised type. Whether general tuberculosis is ever recovered from at all it would be very hard to say. Such a high authority in the matter as Sir William Jenner was of opinion that general tuberculosis is recovered from in children much more commonly than is supposed. It depends a good deal upon what you mean by generalised. No doubt deposits of tubercle in the lungs and abdominal organs may apparently be recovered from; and years afterwards you may find evidence of it in calcification of the glands. But once tubercle has become really generalised, I think anything which you can do in the way of treatment is not likely to influence the process much. The same is true of tubercle of the bronchial glands, which, merely from its situation, it is difficult to treat; for you cannot get at the bronchial glands. Not even the modern surgeon has yet attempted to excise them. For practical purposes, therefore, your treatment in both general and bronchial tuberculosis must resolve itself into simply putting the child into the best possible hygienic conditions, in the hope that its own natural powers of resistance may be able to bring the disease to a favourable termination. In a case of abdominal tuberculosis, on the other hand, you can do a great deal by treatment. There are no cases of tuberculosis which you approach with more hopefulness than those of abdominal tubercle in children; and I should say that the prognosis will be better in older children than in younger, for the reason that in young infants tubercle generalises so fast that you do not often have to deal with the local disease only. In older children generalisation occurs more slowly, so that you have a chance of getting it in time and treating it successfully. The first thing to do is to attend to the general surroundings of the child. Such children should be sent away, if possible, out of towns, and be taken to a place like Margate and spend their time by the sea-shore in a wheeled carriage, lying down. The abdomen should be supported by a broad and firm binder. You will find it an advantage to apply iodoform ointment

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locally; it should be rubbed well into the skin of the abdomen. I also often use, and I think with advantage, liniment of mercury, half an ounce or so being rubbed in night and morning all over the abdomen. Above all, make it your first care to see that the stomach and bowel are in good order, so that the child can take and digest a suitable quantity of food. You must correct gastric disorder if it exists, and do all you can to increase the appetite. If there be diarrhœa you must make a point of checking it before you do anything else. Having done that, you can proceed to feed the child up, seeing in particular that it has plenty of fatty foods. With regard to medicines, you will find help from cod-liver oil, iodide of iron, and creosote. You see there is nothing in this line of treatment other than what your common sense would dictate. But you have often to consider in those cases the question whether you are to continue medical treatment, or whether you are to have recourse to surgical aid. I do not think it is often advisable to have recourse to surgical treatment in those cases right away. I think the necessity for surgical treatment in abdominal tuberculosis in children is very much exaggerated. Taking the last six cases of purely abdominal tuberculosis which have been under my care, simply as they turned up in the out-patient room, and all between three and nine years of age, I had the abdomen opened in only one of them; and that child recovered, although it suffered for a time from fistula of the umbilicus. But the other five children all recovered completely without operation at all. So you see medical treatment is of very considerable use. By recovery I mean that, at the end of two years, some of them now at the end of three years, they are apparently in perfect health. You should speak cautiously of recovery from abdominal tuberculosis. I do not think one has a right to say that a case is completely cured simply because after a few months the symptoms have not recurred. The symptoms in this disease are apt to remain latent for a considerable time. Such a child may, perhaps, get an attack of diarrhœa, or other slight illness, which pulls it down, and, its resistance for the time being lowered, the process flares up again. Seeing, then, that medical treatment gives such good results, what should guide you in having recourse to operation? In the first place, all cases of abdominal tuberculosis should get the benefit of operation if fever and continuous wasting persist in spite of the medical treatment I have sketched out to you. The continuance of such symptoms means that medical treatment is not going to accomplish much, and it is well to give the patient the benefit of operation; although it must be confessed that many such cases, perhaps the majority, do not get well under surgical treatment either. Further, you will always be biassed in the direction of operation if you are dealing with the ascitic form as opposed to the plastic, because the ascitic cases certainly do better under operation than those in which the disease is mainly in the glands or the peritoneum. Numerous statistics bear out this statement. For instance, the records of the general hospital in Heidelberg,* embracing cases at all ages, and not only in children, shows that of forty-one persons operated upon for the ascitic form of tubercle, twenty were completely cured; that is to say, about 50 per cent. Of nineteen cases of the other form, the plastic, only about four could be described as cured. So you see the ascitic form certainly offers a better chance than the other of successful treatment by means of operation. On the other hand, you must remember that the total statistical results of surgical operation are not so much better than the medical statistics if you take them all round, and so it is as well to give every case the benefit of prolonged medical treatment first, especially if there be any objection to operation on the part of the parents. Operation in the ascitic form, properly performed, is almost perfectly safe. In operation on the plastic cases there is a greater risk. Every now and again in such cases, owing to the presence of adhesions, the intestine has been opened into by the operator, and sometimes the bladder also. I have seen the intestine and the bladder both opened into at the same operation. So you will see the procedure is not devoid of danger. Still, if medical treatment fails in such a case, I think it is right that you should have recourse to surgery.

As a substitute for quinine in influenza: ℞ Strychninæ Sulphatis, gr. $\frac{1}{48}$; Cinchonidinæ Salicylatis, gr. ij; M. f. pil. To be taken every four hours. (Packard.)

* 'Jahrb. d. Kinderheilkunde,' vol. liii, p. 766, 1900.

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WITH MR. MARMADUKE SHEILD AT ST. GEORGE'S HOSPITAL.

THERE are a number of cases in my beds of great interest and importance, and really any one of them would serve as a text for a lecture for an entire hour. But perhaps it will be more useful to you if, instead of dwelling upon all the features and points of one case, I spoke to you of several of them, drawing attention to some of the more important points in connection with each of them severally.

HYDROSARCOCELE.

The first case is that of a man, æt. 58, in Hunter Ward, who suffers from that common affection known as hydrosarcocele — an enlargement of the testis, together with fluid in the tunica vaginalis. This will be of additional interest to many of you because it is a very important examination subject, and I fear you all think more of this than what is of utility to you in your future practice. I think it is true that the leading cause of hydrosarcocele, fluid in the tunica vaginalis, associated with enlargement of the testicle, is syphilis, old chronic syphilitic inflammation of the testis. The infection of syphilis may be frequently of very ancient date. In many instances the man has quite forgotten that he has ever had the disease. This man came up from the country, and one would never imagine he had suffered from syphilis. But on going into the history of his past life it turned out that he was a soldier, and he recollected having a sore on the under-surface of his penis, and a rash upon his body. He is nearly sixty years of age, so if he could recollect such events it is pretty strong proof that he must have had a very definite sore and rash. Taking his story into consideration, we did what is usually done in these cases; we evacuated the fluid of the hydrocele by

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puncture, strapped the testis with mercurial ointment, and gave iodide of potassium internally. Now, the large "knobby" enlarged testis is rapidly subsiding. I may mention the case had been diagnosed as cancer of the testis, and the patient had been advised to have it removed. These cases of old syphilitic disease of the testis are very important in practice, and remember that they may occur in any station in life, in any occupation or calling, and at any stage of a man's career. They may occur in members of the highest class in society, as well as among poor hospital patients. When you get a case of chronic enlargement of the testicle to treat, do not be in a hurry to remove the organ by castration before you try the effect of antisyphilitic treatment. This is the most important lesson to learn from a case like the present one.

The next important lesson to learn with regard to hydrosarcocele is the extreme unsuitability of treating such cases by injection. You know hydroceles are still often treated by the injection of various fluids into the sac, such as iodine or carbolic acid. If you carry out this treatment in a hydrocele, which is due to diseased or chronically inflamed testicle, you will do the patient harm and you will not cure the hydrocele; you will exaggerate the already existing disease. Therefore remember, if you ever think of injecting a hydrocele, and you may in your future practice carry out this treatment, be certain there is no disease of the testis before you inject. Remember, also, that hydroceles may be found with other diseases of the testicle. They may be associated with tuberculous disease of the testis, especially in acute cases, and they may, more rarely, be found in connection with sarcoma of the organ. So the condition is not absolutely confined to syphilitic testis, but may, as a more exceptional occurrence, be found in other diseases of the organ.

Fulminating Gangrenous Appendicitis.

The next case I will draw attention to is one of a very different type, but of perhaps still greater importance in practice, because as sure as you sit before me on those benches you will one day be brought face to face with these most anxious and responsible cases. We to-day consider an instance of "fulminating" appendicitis in a little boy æt. 11. First of all let us consider what is meant by "fulminating" appendicitis. Practically this may be defined as a severe inflammation of the appendix, so bad, so septic, that the organ rapidly passes into a state of gangrene in a few hours; and the perforation is associated with septic peritonitis of the most deadly and virulent character. So terrible are these cases that a patient may pass, within twenty-four hours, into a condition of extreme hopelessness, and, indeed, this almost happened to our present patient. I will very shortly read you the particulars of his case. He was a schoolboy, eleven years of age, and was smitten with sudden acute pain rather below and to the left of the umbilicus on the afternoon of October 2nd. Let me interpolate a remark here. It is a very remarkable fact that the pain in the acute fulminating form of appendicitis is often referred to the "pit of the stomach" or the neighbourhood of the umbilicus. Many of you will ask why this apparent contradiction should exist. I would answer the question in this way. The judgment of patients as to the situation of internal abdominal lesions is often erroneous. Nerves irritated near their origins give pain in their distributions, which may be very far from the real site of mischief. In some of these cases where the pain is referred to the region of the umbilicus, I have no doubt that peritoneal inflammation takes place at the back of the walls of the abdomen, and that the inflammation irritates the lower intercostal and sympathetic nerves at their roots, and accordingly the pain is referred over the front of the belly. Also, if perforation occurs, the septic material may be lodged upon, and inflame, a patch of peritoneum a long way from the cæcum. This is especially true with a long appendix. I have even known, in such cases, the inflammatory disturbance to occur on the left side. Such pains often mislead the inexperienced, because they cannot understand how an appendical inflammation can cause pain in quite another part of the belly. The next clinical symptom which was illustrated in this boy's case was this. He vomited several times during the afternoon, and during the night he vomited seven or eight times! The vomiting then ceased and the pain markedly diminished. Acute agonising pain in the belly, associated with urgent vomiting, is a storm signal of the most serious nature, and it usually means that some dangerous lesion has occurred inside. The pain began to cease in this boy after these

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attacks of vomiting. And here is another symptom of immense importance, for nothing is more likely to mislead us than that sudden curious cessation of pain in these terrible cases. It is a most deceptive fact that a patient with fulminating appendicitis and septic peritonitis will lose all pain immediately before death; indeed, he will declare he is much better while the hand of death is literally upon him, and still more so is this the case if the doctor has given morphia liberally! I have on several occasions known a patient with cold extremities, and a pulse which could hardly be felt at the wrist, and a tumid belly, assure me that all his pain had gone and he felt much better, when death was really close at hand! The tympanitis, the rapid small pulse, and the "hollows" under the eyes, should make us aware that the patient is not better, but that the parts are gangrenous and that the end of life is near. With these significant signs in our case the belly was "blown up" and tender, especially towards the right side, with nothing else to be felt. The boy would not tolerate proper local examination. There was nothing noticeable in the temperature; it was only 99°; but the pulse was 104, and "hard." The boy lay in bed with his knees somewhat drawn up. These were the symptoms upon which Dr. Rolleston and myself had to act, and we saw the patient together within twenty-four hours of the first occurrence of the vomiting.

Now, as I say, the seriousness of these cases baffles all description, and they may deceive anybody. We made up our minds on account of the suddenness of the pain, the sickness, and the abdominal distension that perforation had occurred, that the boy had peritonitis, and the question was the situation of the primary lesion. Well, practically, in a young lad there is only one place where you find sudden perforation of intestine, and that is in the appendix. I think it wise in these dubious cases—and they are often very difficult and dubious—to suspect the cause to be in the appendix. We examined the boy's rectum, which it is always right to do in these cases. I have mentioned that the examination was imperfect. We made out merely tenderness. We did not feel fluid or an indurated swelling, which can so often be detected. The two likely areas for perforations to occur in the intestines are the right iliac region and the neighbourhood of the stomach. My own practice in these difficult cases is to rapidly make a small incision, about two inches in length, over the cæcum, and this at once tells you whether the appendix is at fault or not. If you find no lymph, no turbid offensive fluid, no red, inflamed bowel, and the appendix and its neighbourhood be healthy, the incision can be closed by a few sutures in a few moments. The next exploratory incision can be made in like manner over the epigastrium, and the neighbourhood of the stomach, duodenum, and gall-bladder explored. Through one of these openings you will find, in most cases, the perforation, in a dubious case of perforating peritonitis. Well, it was not necessary for us to make a second incision in the case we are considering, because as soon as I cut through the peritoneum with my small incision, in the right iliac fossa a quantity of turbid, offensive fluid bubbled up. And putting my finger down under the cæcum, which was glued to the adjacent coils of intestine by some recent lymph, I soon felt the swollen indurated appendix down in the pelvis. It was "shelled out" with the finger and brought through the wound. This operation was done within twenty-four hours, as I have said, from the first attack of pain. What was the condition of this appendix? It was dark, stinking, and gangrenous! The rapidity with which these appendices fall into death in such cases is almost incredible! It was removed in the customary way, and the boy was treated afterwards in the usual manner,—that is to say, his bowels were opened by small doses of calomel and repeated turpentine enemata; he had an expulsion of gas on the second day, and he began to make what I hoped would be an uninterrupted recovery.* In this I was disappointed. The sepsis in such diseases is very far-reaching and profound. Within two or three days of the operation his temperature began to rise, and he had a slight cough, with difficulty in breathing. This we interpreted as such symptoms are to be interpreted—namely, that he had an infection of the lung or pleura on the right side, or a commencing empyema. The local sounds and signs were those of a patch of pneumonia, which I need not detail to those of you who have had experience in the medical wards. We did very little for these symptoms. We watched the

* The ordinary syringe was not at hand to inject sulphate of magnesia into the cæcum, which I have advised in these cases.

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boy carefully, because in cases of gangrenous appendicitis with lung complications, empyema may ensue, fluid may accumulate in the chest, and when that cavity is aspirated you find it full of sero-pus. This boy fortunately got no accumulation of fluid, and the pneumonia ran through its usual course and gradually subsided. He is now up and out of danger. The operation wound is soundly healed. This is the story, and a very typical story, of fulminating appendicitis in a little boy. I have a strong impression—founded, too, on good experience—that cases of appendicitis in little boys and girls are extraordinarily serious. They are very grave, frequently of the fulminating variety, and rapidly cause peritonitis. I look with particular apprehension upon appendicitis in little children, and I advise you, if in your future practice you get one of these anxious cases and you cannot deal with it yourself, to have a consultation about it without delay. Such cases pass into hopelessness before you know what you are about, and are veritable tragedies. I sympathise very much with the practitioner who has to attend them, because I know how difficult they are to manage, even when one has had long and abundant experience.

Recurrent Attacks of Appendicitis.

Next comes a very different variety of appendicitis, which we will now consider. You can see the case in Winchester Ward. The patient is a woman, æt. 25, who has had three attacks of what is called recurrent appendicitis. She has been smitten with sudden acute pain in her right iliac fossa, great tenderness and rigidity, and a local swelling has been felt, but that has always gone down again. I draw your attention to this one symptom which is very significant—namely, a local tender induration or swelling. This is, in a way, a favourable sign. It shows that the inflammatory process is, at all events, limited. Whenever you can feel a distinct lump in the iliac fossa there is rarely occasion to operate at once, as you would in a case where you estimate that the inflammatory process is diffuse. This girl had typical symptoms. She has suffered from repeated attacks of pain in the right iliac region, and she has also experienced the other signs which are so common in recurrent appendicitis—chronic indigestion, "colicky" pains in the belly, and so on. In such a case, where one attack occurs after another, the usual practice is to operate during the quiescent period. It is well to try what careful attention to the bowels and the digestive functions will do to give such patient relief, with regulation of the diet. But the danger of these cases is, that you never know when another attack will come on; it is all uncertainty. You do not know if such patients will have another attack, or whether the attack, when it does come, will be slight or dangerous. A person who has a damaged appendix practically carries about in his abdominal cavity a source of danger which may plunge him into the gravest peril at any time! It has been frequently said that the unlikely happens in law, and so it does in surgery. A young man who has had one or two attacks has thought very little of them, and is perhaps staying at a house in the north of Scotland, or some other remote part of the country. He may be out shooting or playing at tennis, when he suddenly gets a terrible attack of appendicitis associated with an abscess or peritonitis. A more awkward position no one could be in. There may be no medical man within miles, or at any rate no one who is skilled in operative surgery. So in these cases you have many considerations to think of, and one of the most important is—where is the patient going to live? If he is going to reside in London, or other large town, where skilled surgical aid can be quickly procured, the urgency for operation in recurrent appendicitis is, perhaps, not so great. But when a patient who has had appendical troubles is going to live in a remote place, perhaps one of the Colonies, you should always urge operation very strongly. An operation for recurrent appendicitis may be exceedingly simple, or it may be exceedingly difficult. Those of you who saw this patient operated upon the other day would probably have the idea, as you passed out of the theatre with your hands in your pockets, that these cases are quite simple, and that you could do them any day. But if you come to the theatre and see the operation done often, you will one day observe a case which will tax the resources of the surgeon to the uttermost. It will perhaps take him an hour to get the appendix out. In the case operated upon the other day the customary incision was made, the cæcum appeared in the wound, the appendix, which was found to be swollen,

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club-shaped, and bound down by adhesions, rolled into sight, and was easily removed. The reverse side of the picture is to come down upon a confused mass of inflammatory adhesions and intestine from which you can only separate the appendix with extreme difficulty. I have not yet been beaten by a case, but I have been very nearly defeated. I had such an operation in this hospital last year, in which it took me a full hour to remove the appendix. I was on the point of closing the wound without being able to find or isolate the damaged organ. If you feel inclined, then, to undertake this operation, remember you may commit yourself to a task of very great difficulty. The method of removing the appendix in these cases is a little different from that employed in the case of a sloughing fulminating appendicitis. In the "quiescent" cases the mesentery of the appendix, which contains a separate artery, is first carefully tied. Then the appendix itself is amputated by a regular method. A cuff of peritoneum is turned down, the organ cut off, and the point of section touched with carbolic acid. The stump is tied and the peritoneal coat is pulled forward and sewn with fine silk over the end of the appendix. Thus a small amputation is regularly carried out. Such, then, are remarks upon two cases of appendicitis of a totally different nature.

Cancer of Rectum.

The next case I shall mention to you is also one of very great importance in practice, because it is frequently overlooked in its early stages—namely, a case, in an elderly woman, of cancer of the rectum, which has been treated by colotomy. Her history is the usual one. For about a year she has suffered from bleeding of the rectum after defæcation, and from some difficulty with the bowels. There has been a discharge of slime and, as she herself expressed it, some diarrhœa. Let me draw this conclusion from the text—it is a very important one in practice—that if ever a patient whose bowels have usually been regular begins to suffer from diarrhœa and the passage of blood or "slime," you may conclude almost certainly that he has got some disease of the rectum, and very likely disease of a grave nature. If you ask a student what are the symptoms of cancer of the rectum, he will probably tell you that the patient is constipated. The fact is that above the cancerous obstruction there is often a hard mass of fæces lodged, irritating the mucous membrane of the rectum, and a discharge of mucus comes away stained with fæces. "Spurious" diarrhœa is the leading sign in a case of cancer of the rectum. The next point of importance is that these distressing cases of cancer of the rectum not infrequently occur in young people. It is not necessarily a disease which is peculiar to the aged. I have seen the disease in a boy of nineteen, and in quite a number of persons under thirty years of age. The common and fatal error which is made in practice in these cases is to treat all rectal symptoms as if they were connected with piles. A patient walks into your room and says, "I am suffering from piles." You say, "What do you mean?" He replies, "I have pain, and I pass blood." And if you do what, unfortunately, is too frequently done in practice, give the patient a box of gall and opium ointment and send him away, you will find three or four months afterwards that he has meantime gone to somebody else, who has made an examination of the rectum and found cancer there. But if you follow out the safe rule in such cases—namely, when a patient comes to you complaining of pain or rectal bleeding, wash the bowel out with a copious enema and examine with your finger—you will not fall into that error. If you have diagnosed "piles" in one of your best patients, and it turns out to be cancer, you will fall into the gravest disrepute. This is one of the ways in which reputations and practices are ruined. You have had your warning, and I trust none of you in this theatre will ever omit to make a proper rectal examination where such symptoms exist. This woman was treated for piles, and when she came here she was of course examined, and an enormous growth was discovered in the rectum—a craggy, nodular mass which no one could mistake, and which implicated especially the posterior wall of the rectum. This is a very important point in the case, that the posterior wall was implicated. If the anterior wall were implicated, the septum between the vagina and the rectum, very little good could have ensued from local removal. One of the first considerations in the treatment of a case of cancer of the rectum is this—can the growth be excised and removed, or not? In order to settle such a very important point, remember to do what was carried out in this patient—examine the

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case under an anæsthetic. You do not know what you can do with cancerous growth of the rectum unless you examine in this way. When this woman was put under the anæsthetic I was able to get my finger above the mass and draw it down considerably. It was free from the bone behind, and from the vagina in front. It is a growth which can be removed. Next comes the great question of excision of the rectum; and you know what differences of opinion exist about it. My own experience of excision of the rectum for cancer has not been favourable with regard to after-results. It is all very well at the time, but I have seen a number of these cases suffer from practically chronic incontinence of gas and fæces, especially when slight diarrhœa existed. They constantly made messes in their clothes, or on the pads of wool which they wear so as to lessen the discomforts. Altogether the condition is one of extreme misery. Another bad result which I have seen after excision of the rectum has been the reverse—namely, that the whole parts have cicatrised up, and, in spite of the painful passage of bougies, these people have had chronic intestinal obstruction. I have witnessed those results not only in my own cases, but after operations by some of the best operators in London. I am especially now speaking of private cases which I have had the opportunity of watching. My opinion of excision of the rectum is not very good, unless it is accompanied by colotomy. I believe that the ideal operation for cancer of the rectum is to see that a colotomy is first well conducted and acting satisfactorily, and then the whole bowel below with the growth removed. The patient is then relieved from the constant misery of the discharge of fæces and gas through the great cavernous opening. But in many of these unhappy cases the patients are so feeble that they can hardly bear the secondary operation to be performed. Colotomy was done yesterday in this case, and in three weeks' or a month's time, if the artificial anus is acting favourably, I shall proceed to excise the rectum by Kraske's operation, removing part of the sacrum and the bowel freely from behind. Time does not allow me to describe these operations now, but they are associated with a good deal of hæmorrhage and some danger, and should not be undertaken by anybody who is not used to operating.

Neuro-mimesis of Knee simulating Tuberculous Disease.

And now I come to a case which is the most interesting of any which has been in my wards this year. It is that of a young woman, æt. 26, who is in the Princess Ward, supposed to be suffering from tuberculous knee. I will tell you the story of her case. I was written to regarding her by a medical man of good experience in the neighbourhood of London, who asked me if I would see at my house a young woman with tuberculous disease of the knee. He also told me that so much for years had been spent upon the treatment of her illness, both in medicine and surgery and change of air and the like, that she had completely exhausted the slender financial resources of her family and relatives. If any operative treatment had to be done, he begged me to take the patient into the hospital for the purpose. The appointment was made, and a relative and friend supported the patient on crutches into my room. Her right lower limb was done up in a Thomas's splint, which had been worn for about two years, and her knee was puffy and swollen. Acute pain and swelling had been said to follow a slight local injury. But yet there was one point—and a little thing may turn the scales of diagnosis,—that although the joint was puffy and swollen, there was no local heat, neither was there any obvious thickening of the synovial membrane. In tuberculous knee, if it has existed two or three years, you may expect to find that "pulpy" feeling of the synovial membrane which is so typical of the disease. I have seen many cases of "neuro-mimetic" knees, and I did not think I should ever be deceived by them. But I am bound to confess that I was at first absolutely misled by this case! I examined it very carefully; I agreed in the diagnosis that it was probably tuberculous, and I told the doctor—and I regret I also told the friends of the patient—that the knee would have to be excised, or the girl's thigh amputated. The front door of the house had scarcely shut before it struck me that the synovial membrane was not so thickened and "pulpy" as it ought to be in such a case. It might be possibly a "hysterical" knee after all! So, to save my reputation, I wrote immediately to the doctor, telling him of my afterthought, and that before we operated we would take the possibility

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of "hysterical knee" into consideration. The girl was admitted into the hospital, the Thomas's splint was taken off, and the limb lightly massaged. Then it became evident after a few days that the great swelling of the knee was almost entirely due to the constriction of the bands and bandages about the limb. After these were removed the puffy swelling went down and the outline of the joint became more evident. It was clear that the muscles had greatly wasted, and the limb was cold and congested. Then we put her under an anæsthetic and broke down adhesions, cautiously, both in knee and ankle. This proceeding was followed by no great pain, no local heat or swelling. Massage was steadily persevered in. She now finds herself able to bend the knee nearly to a right angle, painlessly, and has commenced to walk about the ward. I have no doubt the joint will get absolutely well. Time after time such joints have been amputated! I may mention that this patient was seen in consultation by a well-known surgeon to another hospital. He expressed the same idea that I did, that there was a possibility of the knee being "neuro-mimetic." He mentioned how that years ago he knew of such a case under the care of two of the greatest living surgeons in London of that time. They both diagnosed tuberculous knee, both advised amputation, and it was carried out. It was found afterwards, on close examination, that the joint was perfectly healthy! I have known similar errors. Recollect this remarkable case for your future practice. "Neuro-mimetic" joints may deceive anybody. In private practice such ailments are eminently suitable for consultation and deliberation between several men before you act. The reverse mistake, declaring real disease to be hysteria, is a very grave one, and is especially apt to take place in early caries of the spine. These are the cases which bring discredit on the profession by doctors misunderstanding them. The patients get cured by "miraculous aid," or by going to celebrated bone-setters. The case will be trumpeted forth as "another saved from the surgeon's knife." You cannot study and think of a case like this with too great care.

Epithelioma of Lip.

The next case is one of a type which is very common, and by reason of its very commonness I bring it before you. It is one of epithelioma of the lip in an elderly man, with a concomitant condition of the tongue which is sometimes found—namely, superficial glossitis or leukoplakia. The tongue is inflamed and fissured, and covered with dense white patches of epithelium, due to cellular proliferation and papillary development underneath. It is my belief, which some of you may hereafter be able to verify for yourselves, that epithelioma of the lip is far more common in the country than it is in London. The malady is nearly always brought on by the same cause—namely, the constant friction of a rough pipe upon the lip. If it were not for rough pipes and excessive smoking, epithelioma of the lip would be an almost unknown disease. Quantities of men in London smoke short clay pipes, and yet epithelioma of the lip is, in my experience, not so common in London as it is in the country. If any of you hereafter practise in the country, and especially if you happen to be connected with the local hospital, you will find a common operation will be in the person of an old shepherd or gamekeeper, who will come with a cancer of the lip. I remember how often the late Sir George Humphry performed this operation on market days in Cambridge! There are many things in favour of epithelioma being a parasitic affection. These old men in the country irritate and rub their lips, and the parasite of epithelioma gains access. Such a theory is very tempting, if only we could verify the parasite. But I bring the matter before you apart from its supposed origin, because it is the kind of disease which you will all have to treat in the future. The earlier you attack it the better for the patient. If you can find the man when he has got merely a superficial hardness of the lip, with a white dense thickening of the epithelium and a small chronic ulcer, and you excise such a patch freely by a large **V**-shaped incision, you will very likely cure the patient, and he will not get cancer of the lip again. The operation is exceedingly simple; you have simply to cut out a large portion of the lip and bring it together accurately. And if a large portion is cut away the old-fashioned lip pins hold it in position better, I think, than any form of suture. The mucous aspect should be united by horsehair; the pin can be withdrawn on the fourth day. Any glands which are enlarged below the chin—and they are sometimes infected—

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should be removed by a secondary operation, by incision and turning down a large flap of skin and fascia, and dissecting out the whole contents of the submaxillary triangle. This procedure gives very good results, especially in old men. I told you with regard to this particular case that the man has a very interesting condition of tongue, known as "leukoplakia." This disease is produced by the following causes :—Firstly, the "old enemy" syphilis is a frequent cause of these fissures and thickened patches of white epithelium. The next cause is the excessive smoking of strong tobacco. And if you get the common combination of syphilis, and smoking and strong tobacco, leukoplakia of the tongue is exceedingly prone to occur. Another cause of leukoplakia, but more rare, is what is termed glassblower's tongue. It occurs in those men who blow through the hot tubes used in the manufacture of glass, by which means they are constantly burning and irritating their tongues. But sometimes you will see a marked case of this condition, and you will not certainly be able to assign a cause at all. This malady is sometimes called by other names, such as superficial glossitis, or "psoriasis" of the tongue. The importance of the condition is the fact that it is extremely prone to degenerate into epithelioma. It is hardly too much to say that nearly all cases of leukoplakia will become cancerous if they are neglected and left alone,—that is to say, if rough teeth are allowed to rub the tongue and the patients go on smoking strong tobacco. In the case of this old man, we have removed the cancer of the lip, and have absolutely interdicted smoking. These patients frequently beg for permission to smoke "one pipe" or "one cigar," and so on. Do not allow it. I tell them to choose between relinquishing smoking and a dire disease. I hope we shall save this man from cancer of the tongue. Now I ask you to note the treatment I have adopted. The tongue was painful, and I have applied antiseptics and analgesic applications, combined with parolein by means of a brush. The results are very gratifying, for he is able to talk in complete comfort, and his tongue is better than it has been for years. As there is a marked history of old syphilis, he has had large doses of iodide of potassium. His tongue is so much better that now the little white patches will be painted with a solution of cyanide of mercury. Afterwards we shall give him a cleansing mouth-wash, and counsel him against smoking any more; and if he follows this advice, perhaps he will live the rest of his life and avoid that dreadful disease, cancer of the tongue. I ask you to study the local treatment carefully.

Fracture of the Femur below the Trochanters.

The last case I shall refer to this afternoon is that of a man in Oxford Ward, æt. 34, who was run over by a cart and had his femur broken high up, below the trochanters. I draw your attention to the case because it is a variety of fracture of the femur which is associated with the greatest difficulties of treatment. The upper fragment is drawn forwards by the ileo-psoas, forming a prominence in the front of the groin; and the lower part of the shaft is drawn upwards by the hamstring and inwards by the adductors. If you treat this fracture by the long splint, you are very apt not to get good apposition between the fragments. One of the best forms of apparatus is a large Thomas's splint. Bend it well at the hip, and place the limb so that it rests in a flexed position. The next point is to make extension, and this is best done from the lower part of the thigh, the cord and weight passing over a pulley fixed in a post at the bottom of the bed. The fracture joined excellently in the present case. I need hardly add you will employ daily massage of the muscles and parts about the limb. Of course the question will be very prominent in your minds—why should not a fracture of this kind be wired? An extraordinary difference of opinion exists in London about wiring of simple fractures. My objection to advising the wiring of a fracture like this is the difficulty of its performance; it is by no means an easy operation to do, and it is one which would be associated from its position with risks of sepsis. Such an operation might be readily undertaken by a skilled hospital surgeon, but it is not one that the majority of members of the profession can be recommended to undertake. You may get such a case to treat in a farmhouse in a remote country district, and perhaps in an alcoholic subject. Such a case would be a very responsible one. At the same time it would be justifiable in this particular fracture, if it should be associated with very troublesome displacement, to cut down and wire the fragments together. But,

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in my judgment, only a skilled operator should undertake such treatment. To illustrate to you the difficulties which may occur in these fractures close below the trochanter, I may relate to you that I remember this subject was chosen for discussion by the British Medical Association at their meeting in London. It came to the turn of an eminent surgeon in America to be asked how these cases were treated in his country. He said, "In my part of America the matter is very simple: these fractures are not treated; the 'physicians' have given up attending them because of the prosecutions entailed by them and their results." I mention this to you as a little hint, that no cases are more likely to do badly than fractures high up in the femur. With a cantankerous patient and an unscrupulous lawyer, you may get into sad trouble over an accident of this nature. It is one of the forms of fracture which should be treated and watched with excessive care; otherwise you are very liable to be unjustly and severely blamed, and you may even have legal actions brought against you, with all their expense and co-existent worry.

These, gentlemen, are only some of the cases which are in my beds of unusual interest. There are many others of almost equal importance, and no doubt the whole hospital is replete with cases of equal instructiveness. I am only sorry to see the little use to which the majority of students put their opportunities of studying cases in the wards, which is the only way of being familiar with bad cases of disease; and especially in the learning of treatment which hereafter you will have to follow out, if you mean to live by your profession, as well as pass examinations to enter it.

The Treatment of Epilepsy.—H. Schnitzer ('Neurolog. Centralblatt') reports the results of the dietetic treatment of sixteen cases of epilepsy. This diet was recommended by Balint, and consists of 2 gm. of sodium chloride and 3 gm. of sodium bromide added to the food every day. The diet consisted of milk, butter, eggs, bread, and fruit. The results were not particularly satisfactory, although in twelve of the patients the attacks were either absent entirely or were lighter than they had been on the ordinary diet of the institution. Four patients appeared somewhat worse, and there was no improvement in the mental condition of any.—*Philadelphia Med. Journ.*, Feb. 14th, 1903.

CLINICAL LECTURES
ON
DISEASES OF CHILDREN.

LECTURE VII.

RICKETS.

Delivered at the London Hospital, November 11th, 1902.

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GENTLEMEN,—I shall ask you to-day to study with me a nutritional disorder, which has the peculiarity that it is found in children only—I mean rickets On the Continent rickets goes by the name of "the English disease." That is not because it is commoner in this country than abroad; indeed, I suppose there is no place where rickets is commoner than in Berlin. But it is called the English disease because the first accurate description of rickets as we know it now was given by an English physician, Dr. Glisson, whose name will be familiar to you in association with Glisson's capsule in the liver. In the year 1681 Glisson wrote a treatise on rickets, which was then apparently a new disease. He described it as having first appeared in the south-western divisions of England, and having spread thence over the country. He thought it began in the south-western districts because these were mostly inhabited by the more luxurious part of the community. Whether rickets has increased much since that time it is a little difficult to say; but I think one can be pretty sure that it has increased, because all those factors which tend to predispose to rickets are such as are daily becoming more pronounced. One of those factors is that inability on the part of women to nurse their own children which I have already deplored in a previous lecture. Another is the increase in women's work; with the advent of the industrial epoch in the middle of last century, and when factories were established, there opened up at once a great field for the labour of women. This tempted them away from their domestic duties, and from the personal care of their children. That state of things persists, and in many of the large industrial towns rickets is extremely prevalent, for the reason that women go to work in the factories, and their children are left at home

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badly looked after and improperly fed. A third factor in the production of rickets is found in the tendency for the population to be crowded together in large towns, which is such a striking feature of social life at the present day. Now, so potent are these factors, and so prevalent is rickets in consequence of them, that in many towns more than half the children seen in the out-patient room exhibit the disease in a more or less marked degree. So that the dictum pronounced by Sir William Jenner a good many years ago, when he first began to work at Great Ormond Street, still holds good—namely, that "rickets is the most common, and, in its indirect results, the most fatal of all the diseases which peculiarly affect children."

The word "rickets" is derived from the Norman-French term "riquets," which meant deformities, particularly deformities of the spine, or persons who were suffering from such deformities. And it was owing to the fact that one of the main symptoms of rickets is the existence of deformities of bone that this name was first applied to the disease. Glisson, or one of those who wrote with him, proposed as a substitute a sort of pseudo-Greek term, "rachitis." That, however, presents no advantages over the name commonly used, which has at least the merit that it conveys to you one of the main symptoms that cases of rickets present—namely, deformities of the bones. It would be quite wrong, however, to suppose that that is the whole of the trouble. You would have a totally erroneous conception of rickets as a disease if you were to imagine that it consists solely in the production of osseous deformities. You must remember that there is a visceral side to the affection as well, which, so far as effects upon life are concerned, is more important than the mere deformities due to alterations in the bones. As the bone deformities, however, are the most prominent and striking change, we shall consider them first.

The changes in the bones in rickets are of two sorts. There is in the first place an alteration at the point of ossification, the nature of which has been summed up by saying that there is an exaggerated preparation for ossification and a diminished accomplishment of it. That is to say, the preliminary stages of multiplication of cartilage cells go on in an exaggerated degree, and the cells cease to be disposed in those regular rows which are characteristic of normal ossifying cartilage, and become arranged irregularly. The result of this is that the line of ossification becomes thickened, in consequence of which you can observe, clinically, enlargement of the epiphyses. I show you the cast of the wrist of a child who suffered from rickets, in which you can see very well the thickening and enlargement of the epiphyses due to this exaggeration of the preliminary stage of ossification. Such thickening is usually first seen in the ribs. It begins there, or can be earlier recognised there than anywhere else. In consequence there is produced a row of knobs down the sides of the chest, to which the term "rickety rosary" is applied, because the appearance is like a row of beads. Thickening takes place not only on the outside of the rib, but even more markedly on its inner aspect; and it may so press upon the lung that there is a groove of compressed and solidified pulmonary tissue corresponding to the enlarged epiphyses. The specimen I have here shows extremely well such an enlargement at the ends of the ribs just where the cartilage and the osseous part join. Not only are the bones which are developed from cartilage affected in this way, but a similar process takes place in those bones which develop in membrane. Hence the skull bones undergo considerable thickening at the point where ossification is most active. Here I show you the skull of a child which was the subject of rickets; and in it you will be able to feel that in the centre of the frontal and parietal bones there is a considerable degree of thickening. That is one change which the bones show, enlargement at the point where ossification is going on. But they show in addition another general change, namely, a process of softening. The softening is due to the absorption of the mineral constituents of the bone. If you analyse the bones from a case of rickets and compare them with normal bones, you will find that whereas in health there is 37 per cent. of organic and 63 per cent. of inorganic matter, a bone in rickets shows quite the reverse, namely, 79 per cent. of organic matter and 21 per cent. of inorganic. In other words, there has been an absorption or removal from the bone of its mineral constituents, chiefly phosphate of lime. I shall point out to you the significance of this more fully later on, when we come to the theories which have been advanced to explain rickets.

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When phosphate of lime is removed from the bone you can easily see what the consequence must be. The bone bends; and the direction in which it will bend is determined absolutely by the line of chief pressure. Now practically the lines of pressure depend upon the position which the child habitually assumes. In the leg bones, for example, one usually finds that in the femur there is a bend forwards. In the tibia there is a sharp kink in the lower third, such as you see in the specimen before you; or another common deformity is for the tibia to be curved outwards, producing bow-leg. Now these deformities are largely due to the way in which the child sits with its feet tucked under it. It is that which causes the femur to bend forwards and outwards, and causes the tibia to kink forwards in its lower third. In the same way the humerus tends to bend outwards, and so also do the radius and ulna; for a rickety child tends to sit up and lean forward, supporting its weight upon its arms. In this way it is possible to give a mechanical explanation of the direction of bending, and the variations in them depend simply upon the variations in the habitual attitude of the child.

The other group of changes—the visceral—are of even greater importance than the osseous. I might remind you in this connection of another saying of Jenner, that it would be as reasonable to regard rickets as a disease of bones only as it would be to regard typhoid fever as merely a disease of Peyer's patches. The visceral changes consist firstly and chiefly of catarrhs affecting the lungs, stomach, and intestines. Secondly, they consist in a tendency to fibroid change in some of the internal organs, particularly the spleen, with enlargement of that organ, and an increase in the fibrous stroma. The frequency with which enlargement of the spleen is met with in rickets has been considerably disputed. Probably, however, it is not appreciably enlarged in more than five per cent. of the cases,—that is to say, not enlarged to a degree sufficient to enable it to be felt. These visceral changes have this great importance, that they cause, or tend to cause, death much more than the osseous changes. It is largely a matter of indifference to a child whether its bones are hard or soft, unless it happens to be walking about. But it is a matter of serious importance if it is constantly exposed to attacks of bronchitis or a catarrh in the intestines, because these repeated attacks of catarrh subject the child to a great risk of the development of tuberculosis.

Now, to pass to the more clinical side of rickets, it is well that you should recognise certain distinct types of the disease. First of all there is the acute type, which some people call "acute rickets." But this term is one which it is better to avoid, because many cases of infantile scurvy have been described under it. But in spite of that there *is* a group of cases which one can class together as showing a rapid onset of the disease and a great tendency to the development of visceral rather than osseous symptoms, and as being characterised by considerable tenderness of the bones, as well as a great tendency to sweating. Such children sweat profusely about the head, particularly when they are asleep, and also about the trunk—so much so that you will often be able to recognise them in a children's ward by the way in which they kick off the bedclothes. In all those cases I think you will find that the disease has come on rather rapidly. There is a second group of cases, in which the osseous symptoms predominate, and in which the bending of the bones is the most characteristic and striking feature. These are the cases which are likely to come under the charge of the surgeon for deformities of different sorts. Such patients may suffer from time to time from visceral symptoms also, but throughout the course of the disease the bone lesions are those which most arrest your attention. Then there is a third group of cases, characterised by a special tendency to catarrhs. These may be called the catarrhal cases. They are children in whom the rickets is not very marked, but who tend to have constant attacks of bronchitis or diarrhœa, and in them the rickety element is apt to be overlooked. There is a fourth group which one can profitably recognise—namely, those cases in which there is great laxity of ligaments and muscles. I saw yesterday a child who was brought to the hospital with the complaint that it was unable to walk. When a child of three years of age has not yet passed that "walking milestone" which I spoke of early in the lectures, you may take it that it is suffering from one of three conditions: either it has rickets, or it has mental deficiency, or it is the subject of infantile paralysis. This child I am speaking of was not the subject of infantile paralysis, and there was

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no reason to suppose it was mentally deficient; but it suffered from rickets of the type in which there is extreme laxity of ligaments and great feebleness and flaccidity of the muscles. This is sometimes described as the "acrobatic" form of rickets, and I shall show you at the end of the lecture some lantern slides which illustrate it.

All those symptoms, no matter which are most pronounced, tend to come on pretty uniformly at a particular period of life, and I want to impress this upon you, because there is nothing more important than to know what diseases are most likely to happen at a particular age. This is a touchstone by which you can invariably recognise a man who has not seen much of practice from the man who has. The beginner is always diagnosing conditions which the more experienced man is aware do not occur at that age. Thus I constantly find that young infants are sent down from the receiving-room with the statement that they are suffering from rickets. Now anyone who has seen much of disease in children knows that definite signs of rickets are hardly ever observed below the age of six months; the most likely time for it to come under observation is at about eighteen months of age. By that time the disease usually becomes distinctly marked. It is perfectly true that it *begins* earlier than that. Many people assert that rickets is always diagnosable in the second six months of life; but even if it were, it is only by the time the child is a year or eighteen months old that the symptoms are likely to impress themselves upon the mother.

We may now pass on to consider the causes of rickets. I suppose there is no disease of which it has not been asserted at one time or another that it is due to one of two causes—either to a mysterious nervous disturbance, usually described as trophic, or to the action of bacteria. To this rickets is no exception. There have been people who have asserted that it is due to a trophic influence of the nervous system, and there have been people who have asserted that the whole condition is produced by a micro-organism. I shall ask you to dismiss both those theories from your minds as being, in the present instance, quite unproven. To French authors we owe two other theories: one of them is that rickets is an expression of congenital syphilis, and the other that it is due to dilatation of the stomach. Both of those theories are equally chimerical. The first was put forward by Parrot long ago, and the other by his successor Comby, at the infants' hospital in Paris. Neither of them has received much support, except from its author or his pupils. And, indeed, I think there are practically only two theories which have to be seriously reckoned with as likely explanations of the production of rickets. One of those is that the disease is due to defective absorption of lime salts, or to the production of lactic acid in the stomach dissolving away the lime salts out of the bones. This one might call the "lime theory." The importance of considering this theory is that a line of treatment has been based upon it. Lime salts have been administered to rickety children under the vague notion that a deficient supply of them lies at the bottom of the production of the trouble. In criticism of this view it may be remarked that, in the first place, there is no reason to suppose that children who suffer from rickets are getting too little lime in their diet. On the contrary, such children are usually being fed on cow's milk, which contains far more lime salts than human milk does, because cow's milk is designed to build up the massive bones of the calf, and is therefore not likely to be deficient in calcium. Further, nobody has yet shown that the administration of lime salts is of any use in rickets. I would also point out that the inorganic matter of the bone, the calcium phosphate, is dissolved away or removed from the bones of rickets as phosphate of lime. Were an over-production of lactic acid the cause of the mischief, it would be dissolved away as lactate of lime. So the lime theory is falling into discredit, and people are coming more and more to adopt the other theory, which is supported by clinical observation, and also by a certain amount of experimental work—namely, that rickets is due to a deficiency in the diet of fat and proteids. And certainly your clinical experience will soon teach you that the main characteristic of the diet which tends to produce rickets is that it is an ill-balanced one; a diet characterised by an excess of carbohydrates and a deficiency in proteids and fat. Whether it is the absence of the fat or of the proteid which is the most important factor in producing the disease I cannot tell; one only knows that by increasing the amount of fat in the diet you do much good in rickets; and by increasing the proteids you do good also. It is probably, in most

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cases, the deficiency of both of these which has produced the disease. But why it is that rickets should result from such a deficiency no one has yet explained. There is no direct relation, that any one can see, between a deficiency of fat and proteid in the diet and the softening of bones. Yet there is no doubt that animals which are reared upon a diet which is deficient in fat get softening of bones in some way or another.

So much for the theories which have been advanced to account for the production of rickets. I emphasise the last one, because it is by adopting that theory as a working hypothesis that you are certain to be successful in treating your cases. However that theory may stand pathologically, there is no doubt that therapeutics based upon it are justified by results.

Our next point is as to the diagnosis of rickets. That is not usually a difficult matter in a straightforward case. You will be struck first of all by the fact that rickety children are slow in their development, slow in passing at least some of those milestones which I drew attention to in the first lecture. They get their teeth late, their anterior fontanelle closes late, they sit up unaided late, and they walk late; and all these things are due to the bad development and softening of bones. Then you will recognise, usually without any difficulty, the enlargement of the epiphyses, particularly of the ribs, that being the place where the enlargement comes first. It will also be a little marked at the wrists and elbows, perhaps, and there may be some bending of the bones. These are the points you inquire into in the diagnosis of a straightforward case. But there are certain pitfalls in diagnosis to which I want to direct your attention. One of these refers to the rickety head. Rickety children will be brought to you under the impression that they are hydrocephalic. That happens many times in practice. How shall you distinguish the rickety child from the child with a hydrocephalic head? The head of rickets has characteristics of its own. It is a long, square head, which looks as if it had been developed in a box. It is not globular, like the head of hydrocephalus, and if you compare the foreheads of the two you will find that in rickets the forehead goes up more or less vertically in front and at the sides, whereas in hydrocephalus it overhangs the eyes and bulges out above the temporal regions; the top of the rachitic head tends to be flat, in hydrocephalus it is convex and globular. By attention to these points you ought not to have any difficulty in distinguishing a rickety head from a hydrocephalic one. There are certain mistakes which are likely in connection with the spine of rickety children, particularly those who belong to the "acrobatic" group. There is a tendency in such cases towards the production of a prominence in the lower dorsal spine, and they will be brought to you under the impression that they are the subjects of Pott's disease, or tubercular caries. But you will be able to tell a rickety spine from a tubercular one if you remember that one is due to the actual crumbling in of the bone, and therefore cannot be rectified, whereas the other is a mere kinking due to laxity of ligaments, and so can be straightened out. If you hold the child up by its armpits, if the projection is due to kinking it will straighten out, whereas if it is due to tubercular disease it will persist. Another pitfall into which you are likely to be landed is due to the large development of the abdomen in rickety children. Such cases will be brought to you by mothers under the impression that they are the subjects of tubercular disease, and many of them are diagnosed as such and labelled with the term "tabes mesenterica," against which I have already warned you. There are several reasons why a rickety child tends to develop a large abdomen. You know in the first place tha *all* children tend to have relatively prominent and well-developed abdomens. The reason is that they have a large liver and a small pelvis. In rickets both these factors are exaggerated. The liver of the rachitic child tends to be even larger than in health, mainly from fatty infiltration; and the pelvis at the same time tends to be unusually small and to collapse upon itself, so that there is less room in it for the viscera to sink down into. Moreover the badly developed and flabby muscles of the rickety child's abdomen allow distension to take place easily; and such distension is rendered more likely because rickety children constantly suffer from gastric and intestinal catarrh, which leads to the development of flatulence. So there are many reasons why a child should suffer from a large abdomen; and if you recognise the rickety element you will be on your guard, and you will not mistake the enlargement for that due to tubercular disease.

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There is still another pitfall into which you are likely to tumble, and that is in attributing to disease of the nervous system symptoms which are really due to rickets. I mentioned such a case a moment ago, that of a child which could not walk when it was three years of age. The diagnosis in such a case, however, should not be difficult. You will find that a rickety child, though it cannot walk, is still able to use its limbs. As it sits in its mother's lap it will kick its legs about; or if you tickle the soles of its feet it will draw up the legs, which of course it could not do if they were paralysed. In rickets the trouble is merely an inability to walk; whereas in paralysis there is inability to move a particular group of muscles in any way at all.

Lastly, I would emphasise the importance of your being able to recognise the rickety element in many other diseases. For instance, a child may be brought to you who is mentally deficient, but who suffers from rickets as well; and you can do great good in such a case by treating the rickets, although it is impossible for you to improve the mental condition. Again, when we come to speak of the nervous diseases of children we shall see that rickets is a great predisposing cause in many of the functional nervous disorders, such as convulsion, tetany, and laryngismus; and you cannot hope to treat the nervous disease with success unless you treat the rickets, which is the basis of it. Further, in the case of a child who is the subject of repeated catarrhs, bronchitis, or bronchopneumonia, or it may be diarrhœa, it may be of the first importance to recognise that these are simply the symptoms of an underlying rickety condition.

That brings us to the consideration of what the treatment of rickets should be. Granted the theory of its causation which I tried to bring before you—namely, a faulty diet—your first care must be to alter the child's food. But you cannot hope to do that successfully until you have first put the stomach and intestines into a healthy condition, so that it can digest the food when you give it. So in many cases your first task must be to correct any digestive disturbance which exists. You may have to treat diarrhœa, or gastric catarrh, or want of appetite; and having done that you change the diet. And you will change it in this direction: diminish the amount of starchy things, and increase the amount of proteids and fat. Practically that resolves itself into increasing the proportion of cow's milk and administering cod-liver oil. Of course if the child can get plenty of cream you can dispense with the cod-liver oil. But always increase the amount of milk. A child of two and a half years should be getting two pints of milk a day, whereas it may perhaps have been getting only half a pint. The yolk of egg is also very useful in these cases, as it contains many things which the child needs. It contains phosphorus, fat, and organic compounds of iron, in addition to proteid, and I like to give it early in cases of rickets. In very young children it may produce vomiting; but in most cases it is taken quite well. It should be lightly boiled, so that it is still liquid when given; or you may shake it up with the milk and give it in that way. Many of these cases also do well with the addition of raw meat juice to their food; or you may even give them underdone meat scraped down. A rickety child must be mainly carnivorous. Rickets might be said to be due to premature vegetarianism, and you have therefore to increase the proportion of animal food, because animal food is characterised by richness in proteids and fat. About the lime salts you need not trouble; there is no occasion to administer them artificially, for milk contains them in abundance. Nor will drugs help you much, unless you call cod-liver oil a drug. If the child is anæmic, then give iron in addition. Many people give phosphorus, but I am not convinced that it makes much difference. Attention to the diet and improving the child's hygienic surroundings must be your chief lines of attack, because although it is true that bad diet is the main cause, yet there are co-operating factors. Want of sunlight is considered by many to be one of these. The child should have the benefit of the sun if there is any, and attention should be paid to ventilation, bathing, and clothing. For the deformities you may require to have recourse to surgical aid; but it is surprising how they tend to disappear after a time. Bones which have been very much bent may still, without any particular effort on your part, but simply as the disease passes off, become straightened out again in a surprising way.

In order to prevent the deformities from becoming greater, it is sometimes well to see that

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the child is kept off its legs. To accomplish this you may require to tie the legs together, or put on light splints which project beyond the foot. And you will require to keep them on for many months, at all events during the day, until the bones have become consolidated again. But in the very severe cases, where the child is five or six years of age and where there is permanent deformity, surgical means are alone likely to give you much assistance.

WITH DR. JAMES GALLOWAY AT THE POLYCLINIC.

A Case of Sycosis.

This case was sent to me by Dr. Blake. It is in some respects a remarkable one, because recently the patient suffered severely from *sycosis*, and has got well in an unusually short time; he showed the early lesions towards the end of March. I need scarcely remind you that two main types of sycosis are to be distinguished. The first type is due to infection by means of mould fungi—the trichophytic sycosis, and is the variety in which microscopic diagnosis is most important, because from the clinical appearance alone it cannot certainly be distinguished from the other form, the common variety, namely, that due to infection by the pus-forming micro-organisms, which we may call just now, following the fashion, coccogenic sycosis. The latter is a form which is closely allied to impetigo, and shows itself by inflammation of and infiltration round the hair-follicles, producing a pustular or even generally purulent eruption. As far as we can judge at present of our patient by observing the relics still remaining of the disease, this seems to have been an example of the ordinary coccogenic type of sycosis, in which there probably existed mixed cultures of *Staphylococcus albus* and *Staphylococcus aureus*, the *Staphylococcus aureus* being the main factor. The treatment I usually adopt in this type of disease is the employment of the mildest antiseptics we can possibly use. A scheme of treatment which is very often successful is by the application, in the first instance, simply of boric washes or boric fomentations, the latter being especially useful when there is much crusting, as there probably was in this case. I believe that in the first instance boric fomentations were used by Dr. Blake. The reason we wish to use the less irritating forms of antiseptics is this: parasiticides sufficiently strong to destroy the staphylococci deep in the hair-follicles in the positions in which they multiply and produce perifollicular abscesses and inflammations, induration and disease, cannot safely be used. For instance, it is impossible to use perchloride of mercury, and it is impossible to employ carbolic acid strong enough to destroy those organisms without producing at the same time a great deal of inflammation and irritation of the skin; and if in the treatment of sycosis you use an antiseptic which produces independent skin lesions, you will probably end by finding the disease much worse instead of better, on account of infection spreading to the injured areas. Therefore the first point which you must have in mind is to apply to this condition a mild form of antiseptic treatment. I recommend boric acid, either in the form of douches, or washes, or fomentations, followed by the gentle inunction of dilute nitrate of mercury ointment of the strength of the Pharmacopœia, or even more dilute than that. A very efficient application for the second stage of the disease is sulphur in some form or another. Sulphur, either alone, or with the addition of resorcin, which increases the active power of the sulphur, is very useful. Other preparations which also contain sulphur as the active reagent are useful for the same reason. The form which is much in vogue at the present time is ichthyol, but that, I think, acts beneficially only because it contains a certain amount of sulphur in such organic combination that it can be set free in the tissues.

It is difficult to be certain about the actual source of infection in these conditions, and we have to be careful because of the possibilities of libel; but in this case the infection seems to have been due to the patient having come into contact with infectious material in a barber's shop. He was shaved by a barber on March 22nd, and the pustules appeared on the 26th. Dr. Blake informs us that the treatment which was ultimately successful in this case was by means of "glutol," a dry gelatine preparation saturated with formalin, but that formalin in starch jelly made him worse. I am not familiar with the use of this remedy, but I wish to criticise the matter to-day in view of

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the application of formalin, because, as a rule, we have found formalin as an antiseptic to the skin in such cases of very limited service. It shares the bad qualities possessed by carbolic acid, but in an intensified degree. Formalin readily produces dermatitis with desquamation, as also does carbolic acid, and if you use any desquamative application as an antiseptic in the treatment of sycosis you are likely to make the condition worse instead of better. Injury of the skin is produced by removing the superficial protective horny layer of epithelium, and thus the agents in the production of the disease gain access to deeper layers of the unprotected skin.

There are two other varieties of the disease which we must consider as being coccogenic. But they are of a much more inveterate type than this. This is a variety which is closely allied to impetigo, and with it is associated ordinary acute impetigo in many cases. The first of these other two varieties is that which produces, instead of suppuration, a peculiar atrophying inflammation of the hair-follicles; in other words, an *atrophying folliculitis*. The usual type occurs commonly on the forehead, but also on other parts of the body, and is known as acne varioliformis. This form, so far as actual bacteriological investigation is concerned, seems also to be produced by pure cultures of the *Staphylococcus aureus*. When the hairy portion of the face is affected, you soon appreciate that you are dealing with a completely different type of disease, and one in which the prognosis is very much worse. The second inveterate type, the fourth form of the disease altogether, is the one which I have already taken opportunities of mentioning to you in our meetings here, and which was described many years ago by Milton. The name which he gave to it was very descriptive, though we now think it somewhat unscientific, namely, "lupoid sycosis." That name was given to it because the disease produced peculiarly erythematous scar-leaving lesions, which increased in amount, and finally produced a result which was very difficult to distinguish from cutaneous tubercular disease. This disease, so far as we know, is also of pyogenic origin. No other organism has been found in it except the pus-forming organisms, and yet we have in this type of disease a peculiar inflammation of the side of the face, usually associated with a large amount of erythema, in which the erythema advances and then may recede, affecting the hairy scalp, producing a small amount of suppuration and a certain amount of crusting. But as the disease persists, gradually a thick erythematous scar of badly formed connective tissue is produced. It is very important then, when we have a case of sycosis to deal with, to make up our minds which of the four forms it resembles. The trichophytic form is recognisable by microscopic examination; the fungus producing it is usually a large spore form, and it can frequently be treated with good results in a short space of time. This type which we have seen to-day is closely allied to impetigo, which is often concurrent with it. This form is very amenable to treatment. When we have the "atrophying folliculitis" type of the disease, and the type which produced an erythematous and badly formed scar, we are in face of two much more obstinate affections; and the prognosis you will give and the expectations you will have yourselves of the result of treatment will be very different.

THE MYCOLOGY OF THE MOUTH. By K. W. GOADBY, D.P.H.Camb., L.D.S.R.C.S. Eng. (Longmans, Green and Co., London.)

THIS work is a well-illustrated and concise summary of present knowledge of the organisms found in the human mouth, and is written "primarily for the use of students of dental surgery." It contains excellent chapters on the technique of the bacteriological laboratory—the materials needed, the preparation and standardisation of media, and the method of recording observations, etc. The author knows his subject thoroughly, and has studied it both from the clinical and laboratory points of view. The chapters on Bacteria in Dental Caries, in Dento-alveolar Abscesses, and in Pyorrhœa Alveolaris, are of special interest. To the general practitioner the discussion on the toxic element in pyorrhœa will be most suggestive. In the chapter on Pathogenic Bacteria of the Mouth the author enumerates, among others, the *Diplococcus pneumoniæ*, *B. tuberculosis*, *B. influenzæ*, and *B. diphtheriæ*. Of the latter he makes the important note that "the various forms of membranous disease occurring spontaneously in the lower animals are due to other bacteria than the diphtheria bacillus, with the exception of the cat, which has been shown to develop true diphtheria." While the work of others is freely acknowledged, the book incorporates the results of a large amount of important original work, and should take rank not only as a text-book, but as a standard work on the mycology of the mouth.

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A LECTURE
ON
"CERTAIN ABDOMINAL AFFECTIONS."

Delivered at the Medical Graduates' College and Polyclinic, February 25th, 1903.

By W. ARBUTHNOT LANE, M.S., F.R.C.S.,

Surgeon to Guy's Hospital, and to the Hospital for Sick Children, Great Ormond Street.

GENTLEMEN,—It was with a feeling of diffidence that I consented to come and lecture here, as I found that you had already been surfeited with material of far greater interest than I could ever hope to provide.

I think my best course will be to consider with you those things that have given me much food for thought and anxiety rather than to attempt to lay before you any new views.

No cases cause the surgeon more worry or more difficulty in determining their character than those exhibiting the conditions which result from the presence of peritoneal adhesions in varying stages of development. One is struck by the great amount of intestinal disturbance that occasionally results from an adhesion which appears trifling, while most extensive adhesions in other cases apparently produce no harm whatever. Many diseases are discovered from time to time, and are made for a brief space to occupy almost the whole visual field of some surgeons to the exclusion of everything else, a special sense having for the time deprived them of their common sense. This is particularly true of the so-called specialists. I need now hardly remind you of the energy with which the throat expert in the first instance, the general surgeon in the second, and the general practitioner in the third, attacked those masses of inflamed lymphoid tissue which exist in the naso-pharynx of children, and were unfortunately termed adenoids. Giving a name to this condition seemed to deprive the surgeon of all desire to work out the factors that determined its develop-

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ment, and to leave in him nothing else than an irresistible impulse to remove it, and this was often the case whether it caused any obstruction to the Eustachian tube or naso-pharynx or not. I do not for one moment intend to depreciate the value of this operation, which, if performed under suitable circumstances, and if followed by appropriate treatment, is frequently of the greatest possible benefit to the sufferer. What I object to strongly is its indiscriminate application, the operator being unaware of, or not appearing to recognise, the share the enlarged inflamed masses of adenoid material take, both as effects and again as factors which themselves produce effects. Looking at it calmly now, and with a full knowledge of its causal relationship, we recognise the powerful influence of the suggestion conveyed by a name. Though the sequence is interesting psychologically, and though the effect is promoted to the rank of a primary cause, little harm and a certain amount of benefit usually follows this treatment, even when adopted with little discrimination. The patient in the majority of cases is a child whose wage-earning capacity is *nil*, whose time is of little importance, and if by chance death or serious harm should ensue—occurrences by no means as infrequent in my knowlege as some people would suppose,—no one is deprived of support.

Now I will call your attention to another condition, and that is disease of the appendix, its nomenclature, and the operation which its presence suggests, viz. its removal. At the present moment no one can afford to have any pain or tenderness in the vicinity of a line stretching from the right anterior superior spine of the ilium to the umbilicus without being advised, or indeed urged, to have the appendix removed. It may be argued, however, that in skilled hands such an operation is accompanied by very little risk; that though the patient is unable to follow an employment for several weeks, if all goes well no further trouble can arise in the appendix. Unfortunately all operators are not skilled operators, and one hears of frequent accidents. Some few are unavoidable, and will occasionally occur even in the hands of the most experienced. In a considerable number of cases the appendix shows no apparent abnormality at the time of its removal. Unfortunately a certain proportion of patients who have submitted to an operation for removal of the appendix have not obtained freedom from the symptoms that induced them to be operated upon. I feel sure that, as practical men, you will agree with me when I express a strong opinion that a large number of these operations are performed on insufficient grounds, and that the appendix is made responsible for much more than its share. This results partly from want of knowledge and partly from want of care in making the diagnosis. Let us consider for a few minutes the mechanics of the appendix and of the cavity with which it communicates, and let us try to obtain some information on the reasons or causes that produce trouble in the appendix. Later we can consider those conditions which are liable to be mistaken for it.

The cæcum and the ascending colon are to all intents and purposes the cesspool into which the small intestine drains. The appendix is merely a little blind diverticulum opening into what is the most dependent part of this cavity when the sedentary or erect posture is assumed. Owing to our peculiar relationship to our surroundings, to the modified diet of civilised life, and to many other causes, our drainage apparatus is not worked with efficiency. In a large number of people the cesspool has become dilated and larger than it should be; its wall loses its tone owing to the constant strain thrown upon it; and the daily or occasional evacuation merely represents an overflow from this portion of the bowel, and not any complete evacuation of it. I would call your attention to this most particularly, since many medical men are satisfied to assume that because a patient obtains a daily evacuation at a certain period his drainage apparatus is performing its work satisfactorily. This is a fallacy, and we cannot guard against it too closely. I have on many occasions found the cæcum loaded and distended under such a state of affairs, and as many as four large daily evacuations to follow the complete cessation of food, showing that the whole of that material must have been contained in the intestine at one and the same time. I would ask you especially to regard very critically such cases in which the individual spends a long time in emptying the bowel, as showing that there exists an accumulation in the cesspool and that there is much difficulty in forcing out a surplus. On speaking to such a patient the other day who had to be operated on for acute intestinal obstruction, in whom the cæcum and

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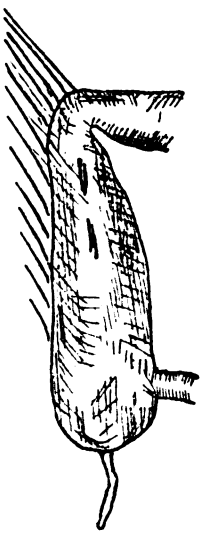
ascending colon were enormously distended, he told me that he always spent at least twenty minutes over his evacuation, and that during that period he passed at intervals small quantities of motion. His friends had on this account conferred on him a new order which they had invented for his benefit—namely, K.P.S., meant to represent Keeper of the Privy Seat. Again I would say, do not be satisfied to desist from investigating fully the condition of the abdomen because the patient tells you that the bowels act with perfect regularity. Satisfy yourself as to the exact state of the cæcum and ascending colon; are they full or are they empty, are the walls thickened, and is the cavity dilated? Is the colon perfectly free from tenderness, or does any pressure on its outline, especially in the right iliac and lumbar regions, cause a feeling of soreness or pain? The investigation of the colon is of vital importance, and should never be neglected by the surgeon.

Let us now consider the conditions that result from an habitual accumulation of fæcal matter in the cæcum and in the ascending colon. It would appear that in performing its function as a cesspool it exhibits a certain difficulty in emptying itself should material be allowed to accumulate in the bowel beyond, or if there exists any mechanical condition depreciating it, such as fat, want of development of the abdominal muscles or of the muscular coat of the bowel, etc. In consequence of the distension the cæcum tends to be displaced downwards and inwards into the true pelvis in the erect posture. Its mucous membrane becomes inflamed, as do also the muscular and peritoneal coats. Peritoneal adhesions form between the outer surface of the cæcum, ascending colon, and the adjoining abdominal wall, which fix the intestine in position and prevent its becoming displaced too far downwards and inwards, or tend to oppose its rotation in such a manner as to obstruct its lumen and produce what is called a volvulus. After a time these peritoneal adhesions become converted into a mesentery consisting of a double layer of peritoneum, which has its attachment in the loin above and outside the hepatic flexure and colon, and which is directed generally downwards, forwards, and inwards, to oppose the drag upon the cæcum in this direction by the material accumulated within the bowel. In many cases the appendix may become attached to the cæcum or to its adhesions, or be retained by them fixed in part, or in its entirety, in an abnormal position. The former frequently produces an abrupt kink in its continuity, the proximal part being fixed, the distal part being free. In any case the fixation of the appendix is often responsible for obstruction of its lumen in a portion of its length, or for the difficulty in evacuating its contents satisfactorily. I believe a very large proportion of so-called abnormalities in the position of the appendix to be due to the peritoneal adhesions about an inflamed cæcum and ascending colon, in some instances the appendix being attached or misplaced at a very early age. For the satisfactory fulfilment of the function of this organ it should be freely mobile



Represents the cæcum, ascending colon, hepatic flexure, and commencement of the transverse colon. [For this illustration we are indebted to the courtesy of the editors of the 'Lancet.']

upon all adjacent structures. Again, the habitual distension of the cæcum produces an inflammation of its mucous membrane, which is most marked at its lowest limit. This inflammation of the mucous membrane may extend to that of the appendix, interfering with its function; or again, this swelling of the mucous membrane at its orifice may block to a varying extent the lumen of the appendix and prevent the free escape of the material secreted in excess by its inflamed mucous lining. I do not believe, except in rare instances, that the appendix becomes inflamed or obstructed apart from accumulations in the cesspool or from conditions

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consequent on, or secondary to, such accumulation. Such tender, inflamed, thick-walled, and often distended cæcums are in my experience exceedingly common, and are frequently, when more acutely affected, regarded as conditions of inflammation of the appendix, or, in other words, as appendicitis. It has happened to me on many occasions to have been asked to operate upon supposed appendical abscesses in which a dose of calomel and a turpentine enema have removed all idea of the presence of an accumulation of pus. Again, I have been requested to remove appendices between attacks where the conditions were clearly due to obstruction of the hepatic flexure of the colon and inflammation of the cæcum and ascending colon consequent upon their habitual distension. In many of these cases the appendix is affected, as one would naturally expect from what I have said; but in a large proportion any trouble there is in the appendix is of a secondary nature, and in a considerable proportion of cases the appendix is perfectly normal. That, however, does not hinder the surgeon from removing it, for he does so feeling that between attacks the appendix may be perfectly normal, and he consoles himself in that manner for the procedure he has adopted.

Next let us see how the formation of peritoneal adhesions and of a mesentery such as I have described to you brings about an obstruction to the free escape of the contents of the cesspool of the gastro-intestinal tract. It occurs in this way. The drag in a downward and inward direction, exerted by the loaded cæcum and ascending colon, produces first adhesions and later a mesentery, along which strain is exerted in an upward and backward direction. The constant exercise of this strain results in a tendency to contraction along the whole length of this acquired mesentery. Its upper portion exerts upon the more mobile flexure an almost unopposed traction, which drags it upwards and backwards and pins it down to the surface of the kidney and the adjacent abdominal wall in immediate relationship to the ilio-inguinal, ilio-hypogastric, and other nerves in the vicinity. The fixation of the flexure in this position frequently produces pain along the distribution of the ilio-hypogastric and ilio-inguinal nerves thatsuggests most strongly some renal condition, while its influence on the kidney in acute attacks of distension is sometimes such that the patient may have during the onset of an acute attack marked urinary disturbance as well. Again, pressure on or percussion over the loin immediately below the last rib causes very considerable pain and tenderness, which has over and over again led the surgeon to regard the whole condition as renal. Till I became aware of all these facts, I, like many others, cut down upon the kidney in the right side because it had apparently given all the conditions, except the presence of blood in the urine, which one was accustomed to regard as renal in origin, and explored the kidney, the ureter, and their surroundings, and was disappointed to find these structures apparently absolutely normal. This disappointment was to some extent alleviated subsequently by the discovery that the patient was freed from all pain apparently by the operative procedure that had been adopted, and that this freedom from pain continued for a varying period. The knowledge of the true causation of the pain by the conditions resulting from chronic constipation or habitual accumulation of an excessive amount of fæcal material in the cesspool, enabled one to explain the mode in which the patient had received benefit by the operation undertaken to expose and explore the kidney. Since the free exposure of the kidney resulted in the displacement forward from its posterior attachment of the new mesentery which controlled the lumen of the bowel at the hepatic flexure in many cases, especially if no effort was made to obviate accumulation in the cæcum and ascending colon, the mesentery, displaced from its attachment at the time of the operation, recovered its old function, and again obstructed the overflow from the cesspool.

The symptoms that people suffering from this condition complain of vary enormously in different individuals, partly with the sex of the individual, the age, the amount of fat, the strength of the abdominal wall, the habits of life, and the disposition of the patient. It may therefore present itself under many guises, and may simulate other conditions. For instance, when the subject is a woman and the abdominal wall lax, it is frequently associated with mobility of the kidney, which produces symptoms closely resembling in many patients those of obstruction of the flexure. Or again, symptoms of movable kidney may be coincident with symptoms resulting

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from obstruction of the bowel, and one or even both may require to be treated surgically.

The similarity in the symptoms presented by a right movable kidney and by an obstructed hepatic flexure are much greater than is generally supposed. They may both give a feeling of dragging in the loin, tenderness on firm pressure below the last rib posteriorly, a feeling of fulness or tenderness or pain in the right iliac fossa, especially towards the end of the day or after the erect posture has been assumed for some considerable time. It may also be noticed on getting out of bed and changing abruptly from the supine to the erect posture. In both cases there are occasional exacerbations of these pains, which may spread to the whole abdomen and may be associated with a feeling of nausea or even with vomiting. Firm pressure exerted from before, backward upon the loin and abdomen on the right side, gripped between the thumb in front and the fingers behind, may cause considerable pain, and if the kidney descends it may be difficult to differentiate the pain and soreness of a mobile kidney from that of the cæcum and colon. Both conditions may be relieved by purgation. By the assumption of the supine position relief is often obtained in both,—in the one case by the relief of the urinary and venous engorgement of the kidney, and in the other by the strain being taken off the newly developed colic mesentery and adhesions, by means of which the lumen of the hepatic flexure is freed and the exit of the contents of the cesspool facilitated. A feature of some assistance in the case of a painful movable kidney is a tenderness of its ureter, which can be best detected by pressure on this structure as it crosses the psoas and the iliac vessels. This, as you will remember, crosses a line stretching from the anterior superior iliac spine to the umbilicus, the so-called McBurney's line. I have known this tenderness to be so marked as to lead to the soreness of the ureter being mistaken for an inflamed appendix, and in consequence of this mistake the appendix was removed without the patient obtaining any but the temporary benefit that resulted from the prolonged assumption of the horizontal posture. This pain and tenderness were permanently removed by nephrorrhaphy. Of course a certain amount of digital familiarity with the varying conditions of the kidney and colon enables the surgeon after a time to differentiate between the subjective and objective symptoms of these two conditions, whether present at the same time or not, and, if both are present, to determine the relative importance of each. I have operated for the conditions of obstruction in the ascending colon and cæcum in every stage of severity, and for the volvulus produced by the rotation of these structures upon a common axis formed by the normal mesentery of the colon and cæcum, and of the newly acquired mesentery I have described. I have exposed an acutely inflamed, distended, and almost gangrenous cæcum and colon, which were so softened as to rupture when freed from the pressure of the abdominal wall. I have operated very frequently in cases at the other extreme suffering from symptoms which were almost typically renal, and concerning which I have already given a sufficient description. Immediate relief has been obtained by freeing the hepatic flexure from the strain upon it by completely dividing the newly formed mesentery and adhesions. If, after this operation has been performed, the capacity of the cesspool be controlled by regular and systematic flushing of its contents, or by massage, there is no tendency for the troublesome mesentery to re-form. But if, on the contrary, this portion of the bowel is again habitually overladen, the new mesentery re-forms as before.

A somewhat similar anchoring of the sigmoid loop develops by the formation of adhesions along its outer aspect. This occurs in cases of cancer of the sigmoid or rectum, producing an interference with the free passage of fæcal matter. I have also on several occasions seen the splenic flexure fixed by adhesions which interfered so much with the function of the bowel as to render necessary their free division. A knowledge of the mode of formation of these adhesions, and of the pinning down of the sigmoid loop, is of great importance in the operations performed for inguinal colotomy, since while it is impossible to attach in the wound a sigmoid bound down by these adhesions, this can be effected with comparative ease if the adhesions be freely and carefully divided, when the mesentery of the sigmoid is restored in a corresponding manner to its original and normal condition. The same condition in a lesser degree exists in the sigmoid as a result of habitual loading of the rectum, or of any cause rendering the escape of fæces difficult,

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such as flexion of the uterus, fibroid or tumour, etc., and may after a time cause considerable local pain and tenderness along the course of the anchored and foreshortened sigmoid. This can only be relieved by active surgical treatment. Should adhesions not foreshorten the mesosigmoid a considerable distension of the sigmoid may ensue. This portion of the bowel may rotate on its axis and produce a volvulus, or twist, which may at any time produce complete obstruction of the bowel. I have resected such twisted loops, establishing continuity in a minimum time by means of the most perfect surgical instrument which I know, namely, Murphy's button. I obtained this instrument, and employed it before anyone in Europe —namely, in January, 1894,*—and during a long experience I have never had the slightest trouble in its use.

The following is an account of one of the operations of resection of the sigmoid for volvulus performed by me, and published in the 'Medical Press and Circular,' May 4th, 1899 :

"Mr. Arbuthnot Lane operated on a case of intestinal obstruction in a middle-aged woman who had been admitted under Dr. Frederick Taylor. Her abdomen was enormously distended; the bulk of the swelling seemed to be due to the presence of a large resonant mass in its interior projecting beyond the adjacent coils of intestine. The patient gave an indefinite history of attacks of previous obstruction, which had been both acute and chronic. Mr. Lane made an incision along the length of the linea alba, through which there at once leaped out a hugely distended coil of bowel; this measured 1 yard in length and 18 inches circumference; the wall of the bowel was exceedingly thick, and its mesentery opaque and scarred. Further examination showed that the condition was one of volvulus of the sigmoid. He adopted the method of excision which he had urged at a meeting of the Clinical Society of London on April 26th, 1895, and resected the whole loop, using the largest size of Murphy's button for the purpose of joining the divided ends, controlling the proximal and distinct portions of the bowel during the process by his own clamps, which are now used pretty universally. It was the largest volvulus that Dr. Taylor or Mr. Lane had ever seen. Mr. Lane said that it seemed to him that this method of excision was the only scientific plan, as he had before pointed out at the Clinical Society in the discussion of the paper on 'Acute Intestinal Obstruction,' by Dr. F. L. Benham and Mr. Silcock. It was quite clear, he asserted, from the condition seen in the case he had just operated upon, that any other recognised form of treatment would not have brought about the complete restoration of function which he hoped might result in this patient. The reasons in favour of excision were, he said—(1) That recurrence is impossible. (2) That a loop which is often acutely inflamed and extensively ulcerated, and the source of immediate danger from perforation, gangrene, septic absorption, etc., is at once removed. (3) That no drainage is necessary, and no fistulous opening remains. (4) That it is a procedure to which no reasonable objection can be offered Button was passed on 31st day, April 12th, 1902." The patient made an uninterrupted recovery.

If a case of volvulus of the sigmoid is seen at a comparatively early date, as is not uncommonly the case now, because of the much greater experience of general practitioners in abdominal conditions, it may be quite unnecessary to excise the twisted loop. The reason of this is that the sigmoid, though considerably dilated, is not sufficiently inflamed but that it can readily recover if material is able to escape freely from it. I have just operated on a case of this sort in which we were able to recognise the presence of a volvulus of the sigmoid from the history and from the symptoms present. There I exposed the loop, turned it out of the abdomen, untwisted it, evacuated its contents, closed the aperture in the gut, and then sutured its enormously hypertrophied outer longitudinal band of muscle-fibres to the edge of the oblique incision in the left groin, which, for this purpose, was made close to the anterior inferior spine of the ilium. Being fixed through a considerable portion of its length along its convexity, the sigmoid cannot again rotate and occlude its lumen. By adopting this method instead of excision I obtained a reasonably good working arrangement, and avoided any such risk as is entailed by the removal of the distended loop. This risk must necessarily vary with the circumstances under which the patient is operated upon.

* 'Lancet,' April 21st, 1894, p. 1006, "Sarcoma in mesosigmoid; resection of sigmoid with growth; use of Murphy's button; recovery."

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Much of the mystery which surrounds volvulus of the cæcum and ascending colon, and of the sigmoid, is due to the fact that the causation of these conditions has not been recognised. It has been assumed that they are acute conditions, and that the symptoms of obstruction are severe and sudden in their onset. Nothing can be further from the truth than this. I have never seen such a thing as a volvulus of either of these portions of the big bowel arising suddenly in a perfectly normal subject. The displacement has existed for a very long time, as evidenced by very definite attacks of obstruction, diarrhœa, flatulence, pain, etc.; the coats of the bowel are enormously hypertrophied; they are also inflamed, showing the presence of adhesions and thickening of the mesentery; and the length and calibre of the bowel has become much greater than normal.

Difficulty in performing its function as a drain-pipe has existed for a considerable time, till finally an excessive loading makes the chronic partial obstruction a complete one when a stoppage occurs.

The sooner we recognise the causation of volvulus and its chronicity, the more readily will we avoid its occurrence, the earlier will we recognise it, and the more successfully will we treat it. By pointing out the causation of the conditions which result from defective drainage, and the mode of their recognition and treatment, I hope I have succeeded in convincing you of the great importance of attending properly to the manner in which the intestinal tract performs its functions.

CASES OF TUBERCULAR DISEASE OF THE CÆCUM, AND OTHER CONDITIONS SIMULATING APPENDICITIS;

WITH CASES OF APPENDIX ABSCESS IN UNUSUAL POSITIONS.*

By FREDERIC EVE, F.R.C.S.,
Surgeon to the London Hospital; and Member of Court of Examiners, Royal College of Surgeons.

Appendicitis and appendix abscess are so common that when a patient is suddenly attacked with abdominal pain and pyrexia, followed by tenderness in the right iliac fossa, especially if there be a swelling in that region, one is at first sight strongly biassed towards the idea that the condition is due to appendicitis.

I propose in the first place to consider a group of cases of tuberculosis of the cæcum, the symptoms of which so closely simulated appendicitis that a correct diagnosis was at least in some instances practically impossible.

Case 1.—In May, 1902, I was asked to see the wife of a medical man in the country with a view to operate on what was believed to be an appendix abscess. She had suffered for several years with attacks of pain in the epigastrium, supposed to be due to gastric ulcer, but there had been neither vomiting nor hæmatemesis. Her illness began suddenly on May 12th, when she was awakened with acute pain in the epigastrium and great tenderness. The pain continued, and on the 14th instant constant vomiting commenced, although she was taking only liquid food in small quantities. On the 15th she complained of pain in the right iliac fossa, and on the 19th a swelling was noticed in the same position. This became more definite during the next few days, and movements of the right thigh gave pain.

On May 28th I operated. The swelling was exposed by an incision over it, and, after separating adherent omentum, a circular indurated contraction of the large intestine was found at the junction of the cæcum and ascending colon. The omentum showed evidence of recent adhesions. There were no enlarged glands in the mesentery, nor any other condition pointing to carcinoma; but had I not previously seen cases of the same kind, a diagnosis of carcinoma would probably have been made. At the present time her husband reports that she is considerably better in health than she was previous to her last illness, but within the last month or two she has had on one or two occasions attacks of epigastric pain. Her great complaint now is that she is getting so stout.

The operation was performed at night with a defective light; otherwise I should have made an intestinal anastomosis. I preferred, however, to wait until she could be brought to town.

Case 2.—A delicate-looking young man, æt. 23 years, was sent to me by Dr. D—, of Aveley, in January, 1901. History:—For two years he had suffered with attacks of pain in the abdomen, at intervals of one or two weeks. The pain

* Read at the Medical Society, March 9th, 1903.

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was felt all over the abdomen, but chiefly round the umbilicus and in the right iliac fossa. The bowels were constipated during the attacks, but there was no vomiting. A distinct indurated swelling, slightly tender, and dull on percussion, existed in the right iliac fossa.

Operation.—The swelling was due to thickening and extreme contraction of the cæcum, which was bound down in the right iliac fossa. The ileum was somewhat distended. The previous symptoms and the condition of the intestine indicated that the patient was suffering with attacks of chronic obstruction. The lower end of the ileum was therefore united by lateral anastomosis to the inner surface of the ascending colon above the contraction, stitching with silk being employed. He remained well until December, 1901, when he contracted smallpox, and although the disease ran a mild course he steadily lost ground after his return home, and died about eighteen months after the operation.

Case 3.—Lydia K—, æt. 22, was admitted to the London Hospital in July, 1902, with the following history:—A month ago she was attacked with sharp pain on the right side of the abdomen, which came on suddenly with vomiting and lasted two days. The pain returned ten days before admission, and she was confined to her bed for six days. The bowels were constipated. On admission a firm swelling was felt in the right iliac fossa, but there was no superficial tenderness. Pulse 66; temperature 99°. I thought it was probably a case of subacute appendicitis, with a mass formed of adherent omentum around the appendix. The cæcum was exposed by the "gridiron" method, and the swelling was found to be due to an extremely indurated and contracted cæcum. The appendix was not found, and there were some enlarged glands in the mesocæcum, one of which was removed and on microscopic examination proved to be tuberculous. I have kept her under observation, and she remains well.

Case 4.—A delicate-looking young man, on whom I had some time previously performed mastoidectomy on both sides, was sent to me by Dr. S—, of Margate, in April, 1902, with the following history:—"Mr. K— had a subacute attack of appendicitis on March 15th, and I attended him for ten days. On April 7th he had a recurrent attack. There was a small, hard mass in the right iliac fossa, which is still tender."

Operation (April 30th, 1902).—The cæcum was contracted, its walls greatly thickened and board-like, and its lower end adherent to the parietal peritoneum. The appendix not being found, this adhesion was cut through, and a small perforation of the cæcum exposed. The appendix itself then came into view. It was adherent, short, and large. It showed no evidence of recent inflammation. The appendix was removed, and the perforation in the cæcum closed with silk sutures, and a gauze drain left in. The progress of the case was uncomplicated, but after leaving the home a sinus remained for a considerable period.

Case 5.—Florence A—, æt. 18, was admitted to the London Hospital October 23rd, 1902. She had suffered with two attacks of abdominal pain localised in the right iliac fossa and commencing quite suddenly. The first attack occurred last August, and the second about six weeks ago. There was no vomiting, but the bowels were constipated during the attacks. In the right iliac fossa was a small, fixed tumour, not painful or tender, and dull on deep percussion. The temperature the night before the operation was 101°.

Operation.—There was an adherent mass in the right iliac fossa, from which omentum had to be stripped. The lower end of the ileum was identified in the mass; it was much thickened and engorged, and its mesentery contained numerous caseous glands. The cæcum could not be differentiated in the mass, and the appendix was sought for in vain. Primary union.

The clinical symptoms of all these cases were remarkably alike. All gave a history of recurrent attacks of pain, sometimes commencing suddenly. One patient had for some years suffered with attacks of abdominal pain, referred to the epigastrium, and supposed to be due to gastric ulcer, although there was neither vomiting nor hæmatemesis. And in the last attack pain was at first referred to the epigastrium, and later to the right iliac fossa. Another patient had attacks of general abdominal pain, chiefly about the umbilicus, and occurring about every one or two weeks. In nearly all constipation existed; while vomiting was very urgent in one case and noted as present in another, but in the majority it was absent. Moderate pyrexia was present in three cases, and may have existed in the others before they came under observation. The chief point in which this group of cases differs

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from appendicitis was in the absence or slight character of the local tenderness. A swelling of firm consistence was observed in every case. The cæcum was usually thickened, indurated, contracted, and bound down by adhesions, but in one instance there was an annular contraction at the junction of the cæcum and ascending colon, and in another the termination of the ileum was also involved. The nature of the disease was for the most part sufficiently obvious by inspection and from the presence of enlarged glands in the mesocæcum or mesentery; but in one or two instances a doubt existed, and in these a gland was removed and found on microscopic examination to be tubercular.

The appendix was found in only one instance; it was adherent, short, and large, but otherwise unchanged.

In no case was there evidence of general tuberculous peritonitis.

Case 3 exhibited attacks more nearly resembling those of subacute obstruction, and the cæcum was found to be extremely narrow and contracted. It was in this case that an intestinal anastomosis was performed with excellent results.

Although in each patient a definite swelling was apparent, I did not expect to find pus in the majority, as the cases were operated on between the attacks, but rather supposed that the swelling was due to matting of omentum around an inflamed appendix.

Case 1 was the most delusive, there having been marked pyrexia, urgent vomiting, the sudden appearance of a tender swelling in the right iliac fossa, with pain first in the epigastrium, later shifting to the iliac region.

The blood was not examined for various reasons. Some cases occurred before this had come into general use, others were submitted to operation at sight.

The absence of leucocytosis characteristic of tuberculous disease would, however, not have assisted in forming a correct diagnosis, for with one exception an abscess was not suspected, and pus was always absent. Sir F. Treves* quotes a case similar to the preceding. The cæcum was greatly thickened, "leathery," and studded with tuberculous nodules. The appendix was apparently normal, but it exhibited tuberculous changes on incision. In this case, however, the patient presented typical symptoms of tuberculous enteritis, *i. e.* wasting, pain after food, diarrhœa, attended by much mucus, flushing, and sweating, with hectic fever.

In a valuable paper dealing with the operative treatment of chronic intestinal tuberculosis, Mr. Mayo Robson* records two similar cases of tuberculosis of the cæcum. In one there was a stricture of the ileo-cæcal valve, and in the other the cæcum was narrowed. Both were treated by excision of the cæcum, with recovery. He states that in one of these cases the disease was believed to be malignant, and it was only on examination subsequent to removal that its true character was discovered.

I should be disposed, in these cases, to rely on the physiological rest of the diseased part, secured by short circuiting. For tuberculosis of the bowels is usually widely spread, and even if the brunt of the disease falls on the cæcum there could be no security that the whole of the active disease was extirpated.

Mr. Mayo Robson himself speaks favourably of the results of short circuiting in a case of multiple tuberculous stricture of the ileum.

In one of my cases the removal of the appendix, and in two others the exploratory laparotomy, appear to have been followed by quiescence of the disease.

I may recall the fact that the absence of leucocytosis would serve to distinguish these tubercular thickenings from carcinoma of the cæcum.

Difficulties in the diagnosis of other abdominal affections from appendicitis may arise owing to variations in the length and position of the appendix itself, vagaries in the position of the cæcum, and finally to displacements of other abdominal organs. The radiation of pain from the iliac fossa in various directions is not uncommon. It may extend to the loin, to the inner side of the right thigh; and when the appendix is situated in the pelvis there will be pelvic pain, frequent micturition, and even dysuria. In one of my cases the pain extended from the right iliac fossa to the right testicle.

It is, I think, rare for pain to be persistently localised to some point at a distance from the right iliac fossa, as in the following case:

* Allbutt's 'System of Medicine,' vol. iii, p. 916.

* 'Lancet,' September 27th, 1902.

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A solicitor, æt. 52, consulted me a few years ago on account of attacks of pain in the left flank of a colicky character, with constipation, from which he had suffered for many years. In October, 1901, while staying at the seaside, he was suddenly laid up with what he described as one of his old attacks, the pain being referred to the left flank and iliac region. After some days a tender swelling was noticed in the right iliac fossa, and the pain was transferred from the left to the right side. A short time afterwards I operated, taking the precaution in the first instance to open the abdomen in the middle line. The appendix, which occupied the usual position, was surrounded by a mass of adhesion. It was exposed by an incision in the right semilunar line, and after removal exhibited the usual appearances of inflammation. Since the operation he has been entirely free from attacks of pain in the left flank.

In another case the pain was entirely referred to the upper part of the right lumbar region. An officer, while on duty on the North-West Frontier of India, had a very severe attack of abdominal trouble, almost terminating fatally, which was diagnosed as appendicitis, but from the history it seemed very doubtful whether his symptoms had been due to this or to hepatic abscess. During the attack his chief pain was referred to the right kidney region, and a distinct patch of dulness could be made out extending from half an inch below the liver margin into the right iliac fossa. There was a resonant area between this patch and the liver dulness. On his return home I operated, finding an appendix at least six inches in length, which extended upwards from the right iliac fossa behind and to the outer side of the ascending colon; its tip was firmly embedded in a mass of adhesions just below the liver and posterior to the hepatic flexure of the colon. The situation of an appendix abscess is almost as variable as the pain, and its variation depends on the same causes. It may occupy any position, from the under surface of the diaphragm above to the right side of the scrotum below. I met with one in a right hernial sac which contained the cæcum and appendix. The abscess had burst into the cæcum.*

The following is an example of appendix abscess immediately beneath the liver and simulating empyema of the gall-bladder:

* 'Lancet,' vol. i, 1899, p. 140.

A marine engineer, æt. 48, was admitted to the London Hospital in January, 1899, complaining of severe abdominal pain referred to the region of the gall-bladder. He had experienced an attack of dysentery whilst in India. For the last ten years he had suffered with almost continuous pain in the right shoulder. He had an attack of abdominal trouble three years ago, and the present attack commenced a week ago with pain in the right hypochondrium, which "doubled him up," and sometimes extended to the back and right shoulder. There was rigidity of the abdominal muscles. An incision was made along the outer margin and upper part of the right rectus, and on the peritoneum being opened a large collection of pus was found tracking backwards to the renal space; this was drained, and subsequently a sinus remained, which extended across the abdominal cavity at the level of the umbilicus. At a later operation performed in March, by means of a free exploration, I discovered the appendix situated with its tip in contact with the under surface of the liver. It was distended with purulent fluid, and contained a concretion. The cæcum was situated much higher than normal. Some time afterwards the patient left the hospital at his own request, with a sinus still present.

In another case a child, æt. 8, was admitted to hospital with abdominal pain and vomiting, and a smooth, tender swelling, dull on light percussion, and situated an inch and a half above and to the right of the umbilicus. An incision over the swelling revealed an abscess just beneath the parietal peritoneum, shut off by adhesions. The history of this case was misleading in that the child was said to have swallowed a metal whistle, which could not, however, be demonstrated by a skiagram. To mention yet another case, a man was admitted with symptoms of local peritonitis and a tender point, with increased resistance, to the right of, and slightly below, the umbilicus. There was no tenderness at McBurney's point. An incision extending from midway between the pubes and umbilicus to a little above the latter revealed an area of purulent peritonitis just below and to the right of the umbilicus, in the centre of which lay the tip of an inflamed appendix. This was removed, and the patient recovered. The cæcum in both these cases probably occupied the fœtal position close to the umbilicus.

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Intra-peritoneal abscesses extending from the right iliac fossa along the outer and posterior surface of the ascending colon, and abscesses extending upwards in the retro-peritoneal tissue behind the colon, are of course quite common. These are not likely to give rise to difficulties in diagnosis unless there be a complete absence of pain and tenderness in the iliac fossa. This difficulty occurred in the case of a girl, æt. 12 years, who was admitted with a small rounded swelling, having a distinct outline, and situated in the right lumbar region at the level of the umbilicus. It did not move on respiration. There had been pain in the right lower abdomen for three days, vomiting for two days, and slight pyrexia. The right iliac fossa was clear. The nature of the swelling was demonstrated by the existence of marked leucocytosis (40,000 per cub. mill.). In one case in which I anticipated an abscess on the outer side of the colon, within the peritoneum but circumscribed by adhesions, I was surprised after opening the abdominal cavity to find no trace of an abscess. On pressing the colon to the middle line I detected a bulging of the outer layer of the mesocolon, and discoloration at one point. A finger thrust through this entered a large abscess within the mesocolon.

Perhaps the greatest difficulty occurs in distinguishing in the female pelvic abscess and pelvic peritonitis, arising from inflammation of an appendix dropping down into the pelvis, from similar conditions originating in pyosalpinx, suppuration of the ovary or ovarian cysts, and parametritis.

In many instances the diagnosis can only be settled by laparotomy. This was my experience in the case of a woman, æt. 22, who was admitted to the London Hospital with the following history:—She had had two miscarriages, the last seven and a half months previously, when she was confined to bed for three weeks. She had an attack of abdominal pain two months ago, and another three weeks later. The day before admission she was attacked with pain in the lower part of the abdomen and right side, and at night vomited several times. Both micturition and defæcation were painful. The abdominal walls were rigid, and there was fulness low down in both iliac regions and also centrally. The uterus was not enlarged; cervix low down and lacerated. Temperature 101·7°. Two days later there was general resistance over the roof of the pelvis, and some fulness in Douglas's pouch. The appendix was not suspected, as it was thought more probable that the pelvic peritonitis was due to inflammatory trouble following the miscarriage.

Operation.—The abdomen was opened in the middle line. A swollen, rotten appendix, about an inch and a half long, was situated in the middle line of the abdomen about the level of the brim of the pelvis. It was surrounded with pus, and the pelvic cavity also contained sero-pus.

This case impressed me more in that I had previously operated on a case of right pyosalpinx in a woman who complained of pain localised in the right iliac region, and vomiting. There was slight resistance with some tenderness in the same part. The diagnosis, however, was settled by the presence of a smooth, elastic swelling in Douglas's pouch.

In contrast to the case of appendix abscess within the pelvis, I may mention an example of suppurating ovarian cyst in which the symptoms were so unusual as to lead to a diagnosis of appendicitis. I saw the patient with Dr. Redfern, of Croydon, with a view to operation. Two or three years before she had a serious illness characterised by acute abdominal pains, swelling in the right iliac fossa, incessant sickness, and high temperature. She subsequently had several less acute attacks, all being ascribed to appendicitis. In March, 1901, Dr. Redfern attended her in a similar condition, and noted pain, tenderness, and swelling in the right iliac fossa, and constipation. In July, 1901, he saw her in yet another attack. I operated after the symptoms had subsided, and on making an incision over the right iliac fossa, failed to find the appendix. The pelvis, however, was filled with a small ovarian cyst completely adherent in all directions, and scarcely rising above the brim. The incision was prolonged inwards towards the middle line, and with considerable difficulty the cyst, which contained pus, was enucleated from its bed and the pedicle tied. The cavity was filled with gauze and drained. She had a rise of temperature and rigor on the second day, but ultimately made a good recovery.

Permit me to call your attention to one other example of pelvic abscess, which may be of interest from the method of evacuation employed. A man æt. 31 was admitted with illness of three days'

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duration: severe abdominal pain, vomiting, and constipation. Dysuria was a marked feature, and there was also pain on defæcation. The abdomen was generally distended, and there was a definite area of tenderness and resistance in the hypogastrium. Per rectum a painful swelling was felt between the bladder and rectum, and the anterior wall of the latter was bulged inwards.

Operation.—Median laparotomy. On separating the adherent omentum from the roof of the pelvis, the pelvic viscera were found to be inflamed and matted to coils of intestine by recent plastic peritonitis. The appendix was seen dipping down into the floor of the pelvis, but its lower end was so adherent as to render its separation impossible. Owing to the firmness of the adhesions it was also found impracticable to reach the swelling in the recto-vesical pouch. Bimanual examination (the gloved right forefinger being in the rectum and the left hand in the pelvis) demonstrated a fluctuating swelling, probably an abscess, in this region. The abdominal wound was closed, the patient placed in the perineal position, and a horseshoe-shaped incision was made across the perineum as for external prostatectomy by Zuckerkandl's method. The incision was deepened between the urethra and prostate in front and the rectum behind until the recto-vesical pouch was reached. On tearing through this a large abscess containing foul-smelling pus was opened. It was irrigated and drained, and the patient subsequently recovered.

Had time permitted I would have called attention to certain other conditions which may simulate appendicitis, especially to calculus, dropsy, or empyema of the gall-bladder when that structure is placed below its usual position; to morbid conditions of the kidney, such as calculus, twisting of a floating kidney; and again, intestinal obstruction by a band in the right iliac fossa, and typhoid fever with or without perforation.

Instances have been recorded in which cancer of the cæcum has been mistaken for appendicitis. The following case, in which I nearly mistook an appendix abscess for carcinoma, may be worthy of mention. A man, æt. 66, had been ill for six weeks with abdominal pain and loss of flesh. No vomiting or constipation; temperature normal. In the right iliac region, and extending from the anterior superior spine to the level of the umbilicus, was a tender, rather elastic, and well-defined swelling, adherent to the abdominal parietes. Operation: I made an incision along the right linear semilunaris, and after cutting through the abdominal wall entered a dense fibrous mass incorporated with the parietal peritoneum, and looking like the sclerosed tissue around a carcinoma. Fearing that if I continued to deepen my incision I might wound the bowel, I determined to ascertain if the disease were really carcinoma by opening the abdomen in another position. This was done in the left linea semilunaris, and on introducing the hand no enlarged glands were felt, nor were there any nodules on the surface of the liver. The wound was therefore closed, and on deepening my original incision I reached an abscess cavity, the walls of which were formed by dense fibrous tissue at least an inch in thickness.

Again, colitis may be mistaken for appendicitis, and *vice versâ*.

A Jew, æt. 48, from Johannesburg, consulted me on account of attacks of abdominal trouble, associated with pain in the right iliac fossa, tenderness, vomiting, fever, and the passage of slime. During the past three and a half years he had experienced three attacks, the last ten months previously, which laid him up for a month. When I saw him there was no melæna, no tenderness over the ascending colon, and the stools were then natural. A physician who examined him with me diagnosed colitis. I operated and found a very unusual condition. The distal portion of the appendix, an inch and a quarter in length, had been entirely separated by ulceration from the proximal portion. It was attached to the ileum near its termination, and on cutting through the adhesion I found a small perforation of the bowel through which the contents of the appendix had discharged themselves. The small opening in the ileum was stitched up, and the patient made an uneventful recovery from the operation.

Gingivo-dental Treatment of Alopecia.—The trophoneurotic theory of alopecia suggests treatment of the jaws and teeth as important factors in its cure. Trémolières reviews recent experience in this line. In all but two out of twenty-one cases attention to the mouth was followed by the re-growth of the hair.—*Journ. A. M. A.*, February 21st, 1903.

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CLINICAL LECTURES
ON
DISEASES OF CHILDREN.

LECTURE VIII.

INFANTILE SCURVY.

Delivered at the London Hospital, November 15th, 1902.

By ROBERT HUTCHISON, M.D.Ed., M.R.C.P.,
Assistant Physician to the Hospital, and to the Hospital for Sick Children, Great Ormond Street, W.C.

GENTLEMEN,—Our subject of study for to-day is that nutritional disease of infancy known as scurvy. This disease used to be called, and is still called by many people, "scurvy rickets." That is an unfortunate term, because I think it is now quite well recognised that scurvy in infants has nothing to do with rickets at all, but that the co-existence of rickets in many of the cases is a mere accident, and due to the fact that the kind of diet which is apt to produce scurvy is apt also to produce rickets. Beyond that I believe there is no direct connection between the two diseases, and it is better to use the term "infantile scurvy" to designate this complaint. The prefix "infantile" is used because it is apparently not the same disease as scurvy in grown-up persons. It is not the same thing as the scurvy which used to be such a scourge on board merchant ships, or as the "land scurvy" which occasionally occurs in persons who live in unhealthy surroundings. It seems to be a disease peculiar to infants, and I am glad to be able to say that, just as in the case of rickets, it has been chiefly made known by the labours of English physicians, and particularly, one is proud to say, by men attached to Great Ormond Street Hospital. The first cases were published by Dr. Cheadle in the year 1878, and it was more fully described by Sir Thomas Barlow in the year 1883. His work on the subject was so exhaustive and complete that very little has been added to our knowledge of the disease since that time, and infantile scurvy is now known on the Continent by the name "Barlow's disease." Before I pass on to describe it, I should also say that in past years this disease used often to be described under the term "acute rickets." When I was dealing with rickets you will remember I told you that there were some cases of that disease which you might call acute, because the symptoms come on rapidly. But that has nothing to do with scurvy, and the term "acute rickets" should either be abolished altogether, or, if used at all, it should be applied to genuine rickets in which the symptoms come on with unusual rapidity.

What are the clinical characters of a case of infantile scurvy? The child will usually be brought to you by the mother with the complaint that it is unable to use one or both of its legs, or that the legs are swelled, or that the child cries when it is touched. These are the commonest things that the mother notices wrong first, and the majority of cases of scurvy which are brought to you will be brought with such complaints as those. When you look at the child you will usually observe that it appears healthy. These children are generally well nourished, they are often of good colour, or, at all events, not conspicuously anæmic; but when the child is being stripped you will notice that it screams a great deal and is extremely apprehensive. One has been able to recognise cases of scurvy in the out-patient room by the fact that the child starts screaming whenever it sees the doctor, because it is so afraid it will be handled, for in that way the tenderness of the bones is aggravated. When you come to examine the child more closely you will find perhaps that one of the legs is kept immobile, that it is slightly flexed at the knee, and that the thigh is rotated outwards. On further examination you will probably find signs of rickets, —that is to say, slight enlargement of the epiphyses On opening the mouth you will observe usually that one or two teeth have come through, because infantile scurvy occurs with quite an extraordinary degree of frequency when the first teeth are being cut. Of the cases I have seen in the last two years, twelve or so in all, by far the larger number were children of eight to ten months old. You will commonly find, then, that one or two of the lower incisors are through, and that the gums around them are swollen and of a livid purple colour. In the upper jaw you may observe that there are teeth about to be cut, and that the gum over these is in a similarly swelled and discoloured condition. When you come to examine the limbs you will probably find on one or both of the lower extremities, usually towards the end of the femur or the tibia, a thickening which is extremely tender

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when it is touched, and which presents a sort of boggy feeling on palpation. You may find further, as minor symptoms of the disease, a tendency to hæmorrhages from the mucous membranes, and particularly the occurrence of hæmaturia. In rare cases the hæmorrhages may take place also into the looser subcutaneous tissues, and particularly into the orbit. These are the clinical signs which you will find when you examine a typical case; and if you inquire into the history you will find almost invariably that the child has been fed in a particular way—namely, on a patent food, usually along with condensed milk. That is why cases of infantile scurvy are as common, or perhaps even commoner, among the children of the upper classes as in the lower, because these patent foods, being expensive, are to a large extent the perquisite of the rich. It is only extremely rarely that the infant is found to have been fed on the breast. I have never seen such a case myself; but I believe a few are on record in which, in some mysterious way, breast milk has produced scurvy. Once or twice I have seen the disease produced in children who were fed simply on boiled cow's milk, but by far the majority of the cases of the disease have been fed upon a patent food combined with condensed milk.

The pathology or morbid anatomy of infantile scurvy is not a difficult matter to explain. It is characterised by a tendency to hæmorrhages, and the seat of those hæmorrhages seems to be determined by the degree of vascularity of the parts. For instance, swelling of the bones is due to hæmorrhage occurring in the deep vascular layer of the periosteum; the hæmorrhage raises the periosteum, producing the swelling, and the pressure of the effused blood on the nerves produces the tenderness. Sometimes hæmorrhage takes place between the epiphysis and diaphysis and causes a complete separation of the former, so that it may come to lie quite loose. Very rarely this process of hæmorrhage goes on to the development of suppuration. Similarly the hæmorrhage round the teeth is due to the fact that the neighbourhood of the teeth is a place which is unusually vascular. That is all one need say about the morbid anatomy. We will discuss immediately the etiology. But I wish first of all to turn to the diagnosis of infantile scurvy, and to mention some pitfalls which it is well for you to be aware of.

In the first place, you will have to diagnose infantile scurvy from *rheumatism*. I say that because I have had at least two cases of infantile scurvy sent to me as rheumatism, in one of which the sufferings of the unfortunate child had been aggravated by the affected limb being painted with iodine. I am certain that this is one of the commonest mistakes made with regard to this disease, that the swelling and tenderness of the bones are regarded as rheumatic in origin. But that is a mistake which you ought never to make, because at the age at which infantile scurvy occurs rheumatism is unknown. Little children below one year of age do not suffer from rheumatism. I do not know why that is, but one knows it to be a fact. So you see this point of differential diagnosis is merely another example of that principle of reasonable probability which I tried to bring before you in a previous lecture—namely, the principle that at certain ages certain diseases are more likely to occur than others. At this particular age rheumatism is not only unlikely to occur, but it does not occur at all.

Another thing which this condition has been mistaken for, and more excusably, is *syphilitic periostitis*. Syphilitic periostitis usually occurs either in children who are much younger than the age at which scurvy occurs, in the form of syphilitic epiphysitis, which I described when dealing with the subject of congenital syphilis, and is a disease seen during the first few weeks of life; or it occurs considerably later in the form of periostitis affecting the long bones, very commonly the tibia, and then the children are distinctly over the age at which infantile scurvy is usually seen. You will find, too, that other signs of congenital syphilis are absent in a case of pure scurvy, and also that in congenital syphilis there is no sponginess of the gums.

Scurvy may also be mistaken for ordinary *epiphysitis*, and that is a fairly excusable mistake which I have known to be made by people who are perfectly familiar with the usual symptoms of both diseases. The only cases in which this error may be made are those in which there is no affection of the gums. But there are other points which will help you. One of these is that the temperature is always raised in epiphysitis, whereas in scurvy it is not usually so to any extent. But one of the best tests of all is to observe the result

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of treatment. Scurvy properly treated gets well almost at once, so that the therapeutic test is of very great value in diagnosis. If you are in doubt, then, whether a case is one of scurvy or not, put the child upon a diet which is suitable for scurvy, and see what happens. If the child has scurvy you will have done right, and improvement will set in at once; if it has not got scurvy you will not have done any harm. In some cases the therapeutic test may be the only one upon which you can depend.*

There is a fourth condition which affects the limbs, and which it is well to be on the look-out for, so that you do not mistake it for scurvy—namely, *infantile paralysis beginning with hyperæsthesia.* Some cases of infantile paralysis are accompanied at their outset by extreme hyperæsthesia of the limbs, so that whenever they are touched the child cries out. But in such cases the age of the child ought to keep you right. Infantile paralysis is a very badly named disease, because it is not a disease of infants; it is a condition which is almost unknown below one year of age. It occurs most commonly between the ages of two and four. In infantile paralysis, too, there will be atrophy or wasting of the limb, and no swelling of it.

The lesions in the mouth and swelling of the gums may be mistaken for other conditions. They may be mistaken firstly for stomatitis, and sometimes there will be no means of telling whether it is a case of mere stomatitis or scurvy except by trying the effect of diet. These mouth lesions are much more likely to be mistaken for the ulcerations which occur in connection with leukæmia. I remember seeing a child about six years of age lying in the ward of a hospital, and by its bed was a bottle of lime-juice. The child's breath was exceedingly offensive, and on examining the mouth there was a large excavating ulcer of one tonsil and ulceration and lividity of the gums; and one was told that the child was suffering from scurvy, and was being treated by lime-juice. But the child did not benefit by the lime-juice treatment. And on examining the blood we found 200,000 white corpuscles, 98 per cent. of which were leucocytes; in other words, it was a case of acute leukæmia. These latter cases are very often characterised by the occurrence of hæmorrhages into the gums, swelling and sponginess of them, and ulceration. And it is well for you to remember, as I shall have occasion to point out to you when I speak of the anæmias of infancy, that acute leukæmia, although very rare in grown-up persons, is not very uncommon in children; and it is very often accompanied by those lesions of the mouth. But here, again, the examinations of the blood will at once settle the matter.

Cases which are characterised by hæmorrhages into the orbit may be mistaken for two conditions: for *sarcoma* of the orbit on the one hand, or for that very rare disease called *chloroma.* In the case of sarcoma there will be usually signs of sarcoma elsewhere, and the therapeutic test—change of diet—will be conclusive. The differential diagnosis from chloroma I must defer until I come to speak of it under the anæmias.

Lastly, I would mention that renal hæmorrhage due to scurvy is apt to be mistaken for *renal hæmorrhage* from other causes. I have known a surgeon cut down upon a kidney in such a case, expecting to find a stone, because the child had been suffering from profuse hæmaturia. He found no stone, and the hæmaturia persisted until it occurred to those in charge of the patient to make a change in the diet. They did so, with the result that the hæmaturia disappeared in the course of two days. So hæmaturia from scurvy is a thing which you may overlook. Some people believe that it is a much commoner symptom of scurvy than is generally supposed, and that you will commonly find traces of blood in the urine in a case of scurvy if you look for them. Of course, in a case of unexplained blood in the urine you should think of the possibility of scurvy. Here, again, I know of no very definite way of making sure of the cause of the hæmaturia, except by having recourse to the therapeutic test—changing the diet, and seeing what happens.

Next I want to discuss with you what the *treatment* of infantile scurvy should be before we pass on to consider its etiology. It was found very early in the investigation of this disease that it could be cured almost at once by ensuring the presence in the diet of fresh constituents, such as fruit juice and vegetables. And the routine treatment for

* I have lately seen a case of infantile scurvy which had been regarded as one of fractured femur, but the history here should prevent mistake.

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you to recommend in a case which comes before you is that the child should be given milk which is raw,—that is to say, unboiled. Also, any patent food which the child is taking should be at once stopped. You should give every day a few teaspoonfuls of orange or grape juice, or, if you like, a little raw meat juice. The child should also be given baked potato. The best way is to bake the potato in its skin, and scrape away the floury part just beneath the skin and shake that up in the milk. You may ask why one should take that particular part and treat it in that way. The reason is that that part of the potato is richest in potash salts. If you put a child who is suffering from scurvy on such a diet, the child will, in the immense majority of cases, get better straight away. There is absolutely nothing in the whole range of therapeutics more striking than the effects which you get from a radical change of diet in infantile scurvy, and there is nothing in regard to which you will deserve or obtain more credit in practice. But on the other hand there is nothing in regard to which, if you fail to recognise the condition, you will deservedly get more blame, because it is a disease which it is quite within the compass of medicine absolutely to cure if it is only recognised.

That leads me on to the discussion with you of the last question—namely, what is scurvy really due to? Why is there this hæmorrhagic tendency? As I have said, it is due in some way to there being something wrong with the diet. The results of alteration in the diet make that point quite clear. What is there, then, in a diet of patent food and condensed milk to produce this tendency to hæmorrhage in the child? That is a question to which no final answer is yet forthcoming; but I think one can get at least near to some idea of what the nature of the fault is by carrying out a series of simple observations. The first thing to determine is whether the fault in the diet is a positive or a negative one. I mean, is there something in the food which should not be there, or does it not contain something which should be there? In other words, is it an error of omission or an error of commission? You can easily settle that in this way. You can leave the diet exactly as it was, and you can add to it fresh fruit juice. If you do that you will find that the child gets well. So it is clear that there is nothing positively wrong in the diet, but that there is merely something absent from it which fruit juice contains. These cases are not due, then, as some people would have you believe, to the development of ptomaines in the food. I do not believe at all in the ptomaine theory as applied to scurvy. Certainly in the case of children any such explanation is quite insufficient. What is wrong, therefore, is that the food of the child does not contain something which it ought to contain. What is that something? Many things have been suggested. Some people think it is a ferment. You can settle whether it is really a ferment by trying the effect of *boiled* fruit juice, for a ferment would be destroyed by boiling. Now I find that boiled fruit juice cures these cases just as fresh fruit juice does. What constituent of the fruit juice is it, then, that brings about the benefit? It must be a "dead" constituent, because it is evidently unaffected by boiling; and it belongs either to the crystalline or colloid group of ingredients. The next step, therefore, is to dialyse the fruit juice and administer the dialysate. If one does that one finds that the dialysate has a curative effect, but the colloid constituents have not. In other words, what is absent from the diet that produces scurvy is a crystalline substance, which is present in fruit juice. One must therefore try the effect of the crystalline substances contained in the juice in a pure form. Of these the citrate of potash and the citrate of lime suggest themselves for trial first. The reason one takes the citrates first is that there is *a priori* reason for supposing that it may be absence of citrates which produces the disease. Cow's milk contains far more citric acid than you might suppose; a quart of it is said to contain as much citric acid as a large lemon. Citrate of lime when it is boiled passes into a less soluble form, and when milk is condensed citrate of lime is apt to separate out in the form of gritty, granular particles in the tin. There are grounds, therefore, for believing that it may be the absence of citrate of potash or citrate of lime which produces scurvy. I have not found, however, that the administration of citrate of potash or citrate of lime in pure form has a definitely curative effect. I have tried citrate of lime in large doses without producing improvement. On the other hand, from a mixture containing malate of potash, citrate of potash, and tartrate of potash, I have found great improvement, which, however, is not brought about so quickly as by the complete change of diet which I have already mentioned. Further than that I have not yet carried my observations, and I cannot speak more definitely. But I think there is ground for supposing that this mysterious alteration in the blood is due to a deficiency of salts of potash, though how it is that this deficiency tends to cause hæmorrhages I am not in a position to explain. This, however, one can say, that the hæmorrhages are not due to any defect in the coagulability of the blood, for in many cases of infantile scurvy in which I have estimated the coagulation-time I have never found it materially different from the normal.

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NOTES ON DIAGNOSIS.

A Post-graduate Lecture delivered by

F. DE HAVILLAND HALL, M.D., F.R.C.P.,

At the Westminster Hospital, December 2nd, 1902.

IT goes without saying that to the medical man engaged in active practice diagnosis is all-important. Unless an accurate diagnosis be made, treatment must be more or less a farce, and any idea of venturing on a prognosis must be put aside. Impressed with the grave importance of the subject, I intend to devote the present lecture solely to the question of diagnosis, and I will base my remarks on cases which have recently been under my care in the Westminster Hospital, or which I have seen in consultation practice. I propose to treat the diagnosis of typhoid fever and gastric and duodenal ulceration, together with the diagnostic value of epistaxis.

The subject of the diagnostic difficulties of typhoid fever came before me on my return from my summer holiday, when I found two female patients in whom the question of typhoid had been raised but not definitely settled.

The first patient, a girl æt. 20, was admitted on the eighth day of illness, suffering from aching pains all over the body and general depression. The illness had started with slight shivering. The bowels were rather confined, and the appearance of the patient was not very suggestive of typhoid fever. There were no spots. On the fourth day after admission Widal's test gave a negative result. The question of pneumonia was raised, but an examination of the blood gave a normal count; certainly no leucocytosis. In the absence of any definite physical signs, and in the presence of a temperature which, when I saw the patient, had for a week been nearly stationary between 103° and 104°, I ventured to make the diagnosis of typhoid fever. This was confirmed by response to Widal's test a week later, and the patient passed through

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an uneventful illness, but with a characteristic temperature, and left the hospital convalescent seven weeks after admission.

This was a fairly easy case for diagnosis after the first week, as by a process of exclusion one came to the conclusion that typhoid fever was the only condition which, in the absence of any physical signs, would explain the patient's persistent high temperature.

Dr. Charles Murchison, who was certainly one of the ablest of the clinical physicians of the latter part of the last century, laid it down as an aphorism that "a fever which in this country (aguish districts excepted) persists beyond seven days and is unattended by any cutaneous eruption, or by signs of local disease in the head, chest, or elsewhere, is enteric fever, even though there be no symptoms directly referable to the intestinal lesion."

This aphorism, though in the main correct, is not so entirely to be depended upon since influenza appeared in the country. This disease has greatly increased our diagnostic difficulties, and especially with regard to typhoid fever. I have seen quite a number of cases which formerly I should have suspected to have been typhoid, but which have turned out to be manifestations of influenza. Hence in the early days of a febrile attack, caution is more than ever required before making a definite diagnosis.

Before the discovery of Widal's reaction there was often no other means of arriving at a diagnosis than by a process of exclusion; now we usually have Widal's reaction to confirm our diagnosis, though it may be absent, even in a typical case of typhoid fever.

The second case was also a girl æt. 20, who was admitted with a temperature of 102° F., with the history of a fortnight's indisposition. She had not had shivering; there was neither diarrhœa nor constipation. The patient had vomited once or twice, but there were no symptoms suggesting the onset of any special acute disease.

On admission the patient complained of pain in the left supra-scapular region. Her breathing was rapid, and pulse over 100. Nothing abnormal was found in the chest. The abdomen was not distended.

Two days later (August 24th) a dull note and feeble breathing were detected at the right base. On exploratory puncture no fluid was found. On the 26th exploration gave again a negative result. On the 28th the abdomen was noted as being rather distended and painful. On the 30th another exploratory puncture was made, also with a negative result. Some doubtful spots were seen on the abdomen.

When I took the patient over on September 8th I found that Widal's test had been tried on two occasions; the first time it gave a doubtful reaction, and the second time it was negative. At the base of the right lung there was impaired resonance with deficient entry of air. The temperature, which since admission had been between 102° and 104°, and had been kept down by cold sponging, was just beginning to oscillate, the morning temperature having been 101°, 99·4°, and 98·6° respectively on the three previous days. In view of the negative result of exploration, and having satisfied myself by X-raying the patient on a screen that there was free movement of the diaphragm and no subdiaphragmatic mischief, I decided to leave the patient alone and await the development of the case. From September 8th the temperature gradually fell, with characteristic morning remissions, and by September 21st the temperature was normal throughout the day, and continued so until the 27th, when a well-marked relapse occurred, which unfortunately terminated fatally on October 8th. On the 6th day of the relapse rose-coloured spots were detected on abdomen and back.

On September 16th Widal's reaction was not given, and on October 6th only a very slight response was obtained.

Permission to have a post-mortem examination was refused. This case illustrates one of the difficulties in the diagnosis of typhoid fever, viz. when the physical signs point to fluid in the chest, which exploratory punctures fail to detect.

I was much impressed by a case in which a subdiaphragmatic collection of pus closely simulated the symptoms of typhoid fever; indeed, it was only by means of the X rays that an abscess was recognised and enabled to be treated surgically.

The longer I live the more numerous are the pitfalls I see into which the unwary diagnostician may fall, and on looking over my note-books I find cases both of commission and omission in the diagnosis of typhoid fever. At times the differential diagnosis between acute miliary tuberculosis and typhoid may be very difficult. I remember,

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for example, being called to see in consultation a man æt. 26 who was taken ill with diarrhœa and violent sickness a fortnight before. The temperature had never been above 101° F., and the pulse varied from 104 to 120. Subcrepitant râles were audible all over the chest. The abdomen was distended and somewhat tender, and the superficial veins were enlarged. There were no spots. The patient had previously had some chest trouble, and there was a phthisical family history. My diagnosis was acute miliary tuberculosis, and my prognosis was consequently of a hopeless character. However, five days later I was called in again, and the case by this time had assumed quite another aspect. The pulmonary signs were less marked, the pulse was less frequent, and the stools had a pea-soup appearance, so that I was compelled to withdraw my original diagnosis and substitute that of typhoid fever. The subsequent course of the case confirmed this view, and the patient made a satisfactory recovery. I have often thought over this case in order to prevent a similar mistake, but there are at times cases in which the ablest clinician will come to grief unless he remembers the shrewd adage of the illustrious judge Sir Matthew Hale, that "Time is the wisest thing under the sun."

What are the diagnostic points which will help us in our endeavour to distinguish between acute miliary tuberculosis and typhoid? One symptom in the case I have just mentioned rather put me off the diagnosis of typhoid, and that was the violent sickness from which the patient suffered. I did not then realise how common sickness was as an initiatory symptom of typhoid fever, and that it might be very severe, and even persist throughout the whole illness.

The points to which I have looked to help me in the diagnosis are :—The previous history of the patient, *i. e.* the history of pleurisy or former lung trouble, enlarged glands, or other signs of a tuberculous diathesis. Then the pulse in acute miliary tuberculosis is more rapid than in typhoid, and is out of all proportion to the temperature, and there is an absence of the dicrotism which is such a marked feature in typhoid fever. There is usually greater irregularity in the temperature in acute miliary tuberculosis than in typhoid, and it is often higher in the morning than in the evening. The pulmonary symptoms are usually much more severe than in the early stage of typhoid, and the breathing is more rapid ; occasionally, however, as in a case recently in King William, the patient suffering from typhoid comes under observation with the symptoms and signs of an acute bronchitis. Cyanosis would suggest acute miliary tuberculosis. The abdomen is more likely to be retracted, and is usually not tender ; though if the peritoneum be much involved in the tuberculous lesion, both these conditions may be absent. The presence of tubercle of the choroid would, of course, be diagnostic of tuberculosis. To sum up, we may say that in the early stage it is often very difficult to distinguish acute miliary tuberculosis from typhoid fever, and that in exceptional cases the disease may run its whole course without the true diagnosis having been arrived at.

Another tuberculous affection, viz. tuberculous meningitis, especially in young children, often gives rise to considerable difficulty in diagnosis. I saw a case in consultation in August, the medical man in attendance—an able practitioner of large experience—had given a diagnosis of meningitis. On going into the particulars I came to the conclusion that the patient, a girl æt. 15, had typhoid fever. When I saw her she had been ill three weeks. Her previous health had been fairly good. The illness began with shivering and sickness, and headache which lasted nearly a fortnight. The temperature had kept between 101° and 103° during the illness. Constipation had been a marked feature, and for four days there was retention of urine. The abdomen was distended, there was slight tenderness in the right iliac region, but no enlargement of the spleen. When I saw the patient she was quite free from headache, but was delirious at times. There was no evidence of implication of the cranial nerves. Pulse 120, temperature 102°. Nothing abnormal to be detected in the chest. Regarding this as a case of typhoid fever, I ventured to give a fairly hopeful prognosis. Three days later her medical attendant wrote as follows :—"You will be sorry to hear that our patient took a sudden turn for the worse on Monday evening (the day I saw her), became more unconscious, and developed a higher temperature. I saw her early on Tuesday, 7.30 ; she then had slight 'Cheyne-Stokes' and was quite unconscious, and sank slowly during the day, dying about 11 p.m. There was a stage of rigidity during the

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forenoon, followed by paralysis; no squint or other cephalic symptom. We could not get a P. M."

Was this meningitis or typhoid? Who shall say? The age of the patient was in favour of typhoid. The case was ushered in with vomiting. This symptom is certainly more constant in meningitis than it is in typhoid fever, but, as I have already pointed out, it may be very severe in the latter disease, and last throughout the course of the illness. The point on which the medical attendant laid great stress was the persistent headache lasting for a fortnight. But in a case which I shall mention the headache continued until the second week. In meningitis headache and delirium may occur at the same time, whereas in typhoid the headache almost invariably ceases before delirium begins. That this sequence occurred in the case in question is another point in favour of typhoid fever. Optic neuritis is not uncommon in meningitis, though it is extremely rare in typhoid fever. The same holds good of squinting and of irregularity of the pupils. As I have already pointed out, tubercle of the choroid is conclusive evidence of tuberculosis.

The pulse in meningitis may be hard, and if soft it is certainly not dicrotous. The distended condition of the abdomen was a point much in favour of typhoid fever, as in tuberculous meningitis the abdomen is usually retracted, though in the final stage of this disease retraction may give place to a tympanitic condition owing to paralytic distension of the intestines.

In typhoid the patient commonly lies on the back, whereas in tuberculous meningitis the lateral position is often assumed, and the patient may be coiled up.

It has been stated that "in typhoid if the bedclothes be pulled down the child is indifferent, but in tuberculous meningitis he resists and immediately draws them up again."

The piercing cry (hydrocephalic) frequently heard in tuberculous meningitis is quite pathognomonic of the disease.

Influenza, which has been more or less epidemic in this country for the last thirteen years, has greatly increased our diagnostic difficulties. Over and over again have I seen cases in which the medical attendant has diagnosed influenza in the first instance, and it has only been the after course of the disease which has pointed out the true nature of the attack. Not that I believe that in all cases a mistake was made; on the contrary I think that in a certain number of cases there has been a genuine attack of influenza in the first instance, and that the typhoid fever followed it—in fact a case of mixed infection. The gastro-intestinal variety of influenza is the form usually mistaken for typhoid. The points in favour of influenza are its sudden onset with high temperature, the severity of the pains in the back, limbs, and eyes, the coated tongue, and the great depression. This form generally runs a rapid course, and by the end of the first or commencement of the second week the temperature has fallen. In some cases, however, the temperature is kept up by a complication such as pleurisy, and then the difficulty in diagnosis is intensified. In the spring I saw such a case, and to be on the safe side I advised that it should be treated as typhoid fever; but a negative response to Widal's test, coinciding with a fall in the temperature and a healthy action of the bowels, speedily negatived the idea of typhoid fever. I am sure that the right course in these cases is to take the friends of the patient into your confidence and to point out to them that at an early stage the differential diagnosis is often impossible, and that this being so, the patient should be treated as though he had typhoid fever in order to avoid any risk. If in the course of the second week of the illness the temperature comes down and Widal's test gives a negative reaction, typhoid fever can be excluded, and the patient ordered a more liberal allowance of diet. In some cases, especially in children, the onset of typhoid fever is accompanied with so much bronchitis that the true nature of the attack is liable to be overlooked. I have recently had a case of the kind in King William Ward. He was a boy æt. 10, and was admitted with all the signs of an acute bronchitis. He has had a very severe attack, and when convalescing a condition resembling cancrum oris occurred in the mouth.

The recognition of the fact that bronchitis may be the initial symptom of typhoid fever is usually sufficient to enable a correct diagnosis to be made. The continued high temperature should alone suffice to indicate that the patient is suffering from something more than an acute bronchitis.

At times the mode of onset is very puzzling. Take for example the case of a young lady æt. 12½ years,

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to whom I was called in consultation on January 7th of this year. On the 3rd she came up to London to have two teeth extracted under ether. Unfortunately she came round from the anæsthetic before the second tooth was out. The same evening her temperature was found to be 102·3° F. The next day the temperature ranged between 103° and 105°, on the 6th it reached 106°, and the day of my visit the temperature fluctuated between 102° and 104°. The pulse was 108; there was a soft, mitral, systolic murmur. Nothing abnormal was to be detected in lungs or abdomen. There were no spots, no headache, and no pains anywhere. The girl looked remarkably well, and it was difficult to believe that she was seriously ill. I suggested typhoid fever, ulcerative endocarditis, and neurotic fever as the three most likely causes of her condition, and I noted in my case-book that I regarded the first as the probable cause.

I saw the patient three days later. The temperature had kept up to the level of the first three days of illness, the abdomen was now tumid, the bowels were loose, rose-coloured spots had appeared, and Widal's test reacted. The diagnosis of typhoid fever was therefore confirmed. I again saw the patient three weeks later; she had had a very severe attack, but was doing well until two or three days before my visit. The temperature had shown morning remissions with a downward tendency, but this fall was checked; the temperature again began to rise, and the patient had a sharp relapse, but eventually did well. It was hardly possible to believe that the typhoid fever started on the day the patient came up to London to have the teeth extracted, and on further inquiry it came out that the girl had not been feeling very well for some days before her visit to the dentist, but abstained from saying anything about it lest it should be thought that she was anxious to shirk the visit.

The next case to which I will direct your attention was also a difficult one at the onset in which to give a definite opinion. A youth æt. 20, by profession a musician, who had been much overworked, was taken ill with pains in his limbs, very severe headache, intolerance of light, and a rise of temperature. At the onset a diagnosis of influenza was made. As the headache persisted and the temperature continued high, I was asked to see the patient at the end of the first week of illness. When I saw him the headache had somewhat abated, but was still severe. A few doubtful spots had appeared since the morning, and shortly before I arrived slight epistaxis had occurred. The discs were normal. Nothing abnormal was found in chest or abdomen. I congratulated myself that I had not been called in twenty-four hours earlier to see the patient. Then I do not think that it would have been possible to make a diagnosis; but when I saw the youth the headache was less intense, a few doubtful spots had appeared, but most important of all, there had been nose-bleeding; so that I ventured upon a diagnosis of typhoid fever. I saw the patient again four weeks later, and found that he had passed through a very severe attack of typhoid, and that he was then suffering from pleural effusion. He eventually made a good recovery. In doubtful cases of fever the presence of epistaxis should certainly incline the balance in favour of typhoid. It has been stated that this symptom occurs in 40 per cent. of the cases of typhoid fever, but whatever be the exact percentage, I regard it as a valuable corroborative sign, and it has several times been the determining factor in leading me to make the diagnosis of typhoid fever. That too much stress should not be placed on this symptom is shown by a case mentioned by Fagge and Pye-Smith, in which there was repeated epistaxis and the pulse was dicrotous. A diagnosis of enteric fever was made; but at the necropsy miliary tubercles were found in the lungs and other parts, while there were no appearances characteristic of enteric.

Profuse sweating accompanied by an abundant eruption of sudamina may lead to some difficulty in diagnosis unless the medical attendant is aware of this occurrence. I saw one such case some years ago, and I was able to help the doctor in attendance to a correct diagnosis. The other symptoms of typhoid were fairly well marked, so that there really was no difficulty in deciding what was the matter with the patient if only the sweating and sudamina were put aside as of no diagnostic importance.

Among abdominal affections appendicitis is one which may be confounded with typhoid fever. I have seen several cases in which the question of typhoid has been raised in cases of appendicitis, but there has not generally been much difficulty in arriving at a correct diagnosis. Fortunately the mistake of confounding appendicitis with typhoid

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fever is more likely to be made than the converse of mistaking typhoid fever for appendicitis. It would be a grievous blunder to call the surgeon in to operate on a case of supposed appendicitis and then to find that one had to deal with typhoid ulceration of the cæcum. In the ambulatory form of typhoid fever perforation may occur before the patient has been under medical observation, and in a case of this sort the perforation might very reasonably be set down to ulceration of the appendix. The chief means of arriving at a diagnosis is by investigating the mode of onset: in typhoid fever this is usually gradual, there is a feeling of malaise, headache, and pain in limbs; in appendicitis, on the other hand, the invasion is more sudden, vomiting is more common, and pain and tenderness in the right iliac fossa are early symptoms.

I have already referred incidentally to the difficulties attending the diagnosis of typhoid fever from pyæmia when speaking of a patient in whom the chest was explored three times with a negative result, and also in connection with a case of subdiaphragmatic abscess. In cases in which the suppuration is deep-seated there may for a time be considerable doubt as to the exact nature of the illness, but the greater variation in the temperature is suggestive. It is, to say the least, very unusual for the temperature in typhoid fever to touch normal before, at the earliest, the third week, whereas in pyæmic conditions there may be a normal or even subnormal temperature in the morning with a high evening temperature.

It will be noticed that I have said nothing about the diazobenzol reaction of the urine. Though this reaction is usually present even in mild cases of typhoid fever it is more useful as a negative than a positive test, as it is also occasionally present in pneumonia and tuberculosis, diseases which are not unlikely to be confounded with typhoid. Since the introduction of Widal's serum test I have not employed the diazo test.

A case of hæmatemesis occurring in a woman recently in Queen Adelaide Ward has afforded me an opportunity of considering the diagnosis of duodenal as opposed to gastric ulceration. On admission, owing to hæmatemesis and a history of pain and vomiting in the past, it was taken for granted that the patient was suffering from a gastric ulcer; but after a day or two, when she was in a better condition to answer questions, I had some doubt as to whether the ulceration was not rather in the duodenum than in the stomach. The usual signs of a gastric ulcer—pain after food and vomiting—were present, though not so persistent as is usually the case in gastric ulcers. The point which was in favour of the duodenal ulcer was the large amount of bleeding by the bowel. On one of my visits to the ward I noticed that the patient looked much more blanched than on the previous visit, and her pulse was feebler and more frequent. There had been no return of the hæmatemesis, but on examining the stool I found that it was very copious and tarry, evidently due to altered blood. The insidious nature of the hæmorrhage in these cases is apt to cause it to be overlooked.

I was recently called down into the country to see a young lady æt. 26, and the history I obtained was that on getting out of bed one morning she felt faint and giddy, and fell to the ground. Soon after she complained of pain in the epigastrium and feeling of nausea, and then vomited some dark fluid. Later on she had another giddy attack, and again fell. Next morning she passed a copious black motion. Two days later she became jaundiced, but the yellow coloration had almost completely passed off when I saw her, eleven days after the commencement of the illness. The jaundice I attribute to extension of catarrh set up by the ulceration of the duodenum to the common duct. This symptom has been described as a result of duodenal ulceration. Indeed, Wilson Fox regards the occurrence of jaundice as in favour of a duodenal ulcer against one situated in the pylorus. I look upon the sudden occurrence of giddiness and faintness followed by anæmia in a person who has had indefinite dyspeptic symptoms as very characteristic of duodenal ulceration with hæmorrhage, and to clinch the diagnosis the stools should be carefully inspected.

I have since heard that the patient I have just referred to died on the fourth day after my visit. Her medical attendant writes, "You saw Miss P— on the Monday. There was a slight improvement on that day, but on the next the prostration and occasional vomiting returned. On Thursday morning I found all her symptoms aggravated, her pulse became much weaker, and she died at two

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o'clock on Friday morning. I thought it probable that before I saw her on the Thursday morning she might have had another internal hæmorrhage, but as she neither vomited nor passed anything from the bowels I was unable to verify this. I pressed for a P.-M., but could not obtain one. I signed the certificate as duodenal ulcer, but confess that I did not feel very satisfied about it, although I suppose that it was the most probable cause of all her symptoms."

The differential diagnosis between ulcer of the stomach and ulcer of the duodenum is usually a matter of the greatest difficulty. Pain, which is the most constant feature of gastric ulcer, and in the absence of which gastric ulcer can hardly be diagnosed, is not so commonly met with.

As in gastric ulcer, it is induced by taking food, but generally comes on rather later,—that is to say, it does not usually occur earlier than half an hour, and may be postponed until two or three hours after the meal. Curiously enough, the time of occurrence of pain may be influenced by the size of the meal—the greater the amount, the earlier the pain comes on—so that patients will say that after dinner the pain comes on earlier than after breakfast. The pain and tenderness are in some cases limited to the right hypochondrium, but they may be felt at the epigastrium or umbilicus. Vomiting, another almost constant symptom in gastric ulcer, is frequently absent in ulcer of the duodenum. Hæmatemesis is comparatively rarely met with in duodenal ulceration, while it is very common in gastric ulceration. On the other hand, melæna is more common in the duodenal than in the gastric affection.

One feature in ulceration of the duodenum which is very important is the latency which is sometimes exhibited; this is much more markedly the case than in the corresponding gastric affection. The first example of the kind which came under my notice was one reported by my colleague Dr. Allchin to the Medical Society on October 21st, 1878. The patient, a clerk æt. 25, had consulted Dr. Allchin on his way home from business, complaining of some dyspeptic symptoms and a little colic. There was no elevation of temperature, no increased frequency of pulse, and the man was relieved by pressure in the epigastrium. A simple carminative mixture was ordered, but to Dr. Allchin's astonishment he had a message next morning to say that the patient had died suddenly. At the necropsy a clean-cut ulcer in the duodenum, with thickened edges, large enough to admit of the passage of a cedar pencil, was found, and close by was the seat of a commencing ulcer. In a case of this kind I do not see how it would be possible to arrive at a correct diagnosis. If the patient is seen after perforation has occurred there is usually no indication as to what organ has perforated, but in adult males the possibility of the existence of a duodenal ulcer should always be borne in mind.

In contra-distinction to the latency exhibited by the case I have just mentioned is the case of a gentleman æt. 50 whom I saw last year. The patient had been ailing for two years, suffering from water-brash and vomiting. Later on he vomited several clots of blood, and had tarry motions. I advised abdominal section, but the patient did not consent at the time, and, as his medical attendant informed me, he made a good recovery. However, during the present year he had repeated hæmorrhages, so that he at last consented to an operation, and this was done in a free interval,—that is to say, when the patient was apparently free from all symptoms. The pylorus was found much puckered from previous ulceration and thickened. The patient is now eating well, taking ordinary food, and is rapidly putting on flesh.

I have already alluded to the assistance in diagnosis furnished by epistaxis in typhoid fever. I have recently had in the wards a case in which the occurrence of epistaxis clinched the diagnosis in respect to another disease. The patient was a man æt. 41, who was admitted into the hospital with a history of having vomited blood. On admission he vomited several pints of blood, bringing up about half a pint at a time.

In this patient there was great difficulty in deciding as to the cause of the hæmatemesis. In the bulk of cases the diagnosis has to be made between gastric ulcer and mechanical congestion of the stomach due to cirrhosis of the liver. It is just in these cases that the occurrence of epistaxis occasionally comes to our assistance in making a definite diagnosis. Epistaxis appearing for the first time in an individual over forty should always be regarded with suspicion. I cannot put this view of the question more clearly before you than in words of the late Mr. Prescott Hewett in his

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Presidential Address at the Clinical Society in. 1873.

"Severe epistaxis spontaneously occurring after the middle period of life. What, apparently, could be of so little importance clinically? How little thought, commonly, would be bestowed on nose-bleeding under such circumstances, and yet it is ofttimes the little cloud no bigger than a man's hand." He then quoted the case of a healthy-looking middle-aged gentleman, temperate in all things, no marked disease in any one of his organs; the only thing detected was a little thickening of the radial artery at the wrist, the coats of which vessel were somewhat rigid. "This was all, but it was quite sufficient to lead to the inference that the minuter arteries in different parts were in the same condition as, probably in a more advanced state of degeneration than, the radial."

In the course of three or four years the nose-bleeding recurred from time to time with more or less violence; then there was a slight outpour of blood in the brain, from which the patient recovered; eighteen months later extensive bleeding in the brain, and death in a few hours. This case illustrates one of the causes of epistaxis occurring at or after the middle period of life. Quite recently I saw in consultation a lady æt. 78, who had from time to time suffered from severe nose-bleeding. I was called in on account of profuse hæmaturia. On examining the patient I found her with a pulse of extremely high tension; the area of cardiac dulness was increased, the impulse outside the nipple line, and of a heaving character. The urine, which fortunately happened to be free from blood at the time of the examination, was of low specific gravity, and contained a trace of albumen. The evidence, therefore, was conclusive that the patient was suffering from chronic interstitial nephritis, and that the hæmorrhage from the nose and kidneys was due to a common cause, viz. a degenerate condition of the walls of the arteries, with high tension. To the occurrence of hæmaturia in cases of granular kidney, Sir William Gull very aptly applied the term "renal epistaxis." The hæmorrhage in these cases is usually an effort of nature to relieve undue plethora, and does not require to be checked. In the case I have just mentioned astringents had been ordered; but, fortunately for the patient, drugs have but little influence in controlling hæmorrhage from the kidneys. The appropriate treatment is, of course, to lower the undue tension by careful dieting, aperients, and drugs, such as iodide of potassium and nitrites.

The subject of epistaxis has, however, led me to digress. I was speaking of a case of gastric hæmorrhage which presented difficulties in diagnosing the cause thereof, in which the history of epistaxis materially assisted in elucidating the cause. I have already mentioned that epistaxis may result from a degenerate condition of the blood-vessels such as is met with in chronic renal disease. Another not uncommon cause is cirrhosis of the liver. Given, therefore, hæmatemesis, with a history of previous epistaxis, the presumption is strongly in favour of cirrhosis as the cause of the hæmorrhage rather than gastric ulcer. But in this connection it must be remembered that all blood coming from the nose is not true epistaxis; in some cases the nasal passages act as mere conduits, the blood coming from some more or less distant organ. In profuse hæmatemesis, for instance, the violence of the vomiting may drive some of the blood through the nostrils, and may thus simulate epistaxis. In these instances the blood usually pours down both nostrils, whereas in true epistaxis it is much more commonly unilateral. Soon after the patient whose case I have just referred to was discharged, another patient was admitted suffering from epistaxis. He was a man æt. 49, who stated that his nose had commenced to bleed about a fortnight before his admission. He must have lost a very large amount of blood, as he was very pale, his mucous membranes were anæmic, and his pulse very feeble. This case was a characteristic example of the "site of predilection" of nasal hæmorrhage, as on examining the left nostril an abrasion of the mucous membrane of the septum was found about half an inch from the anterior margin. At first I was inclined to regard this case as an example of epistaxis due to granular kidney, as on admission the urine contained a trace of albumen; but as this disappeared later on and there were no signs of cardiac hypertrophy I rather doubted the accuracy of my original diagnosis. This case illustrates the ease with which epistaxis can often be checked when it is recognised that in a considerable proportion of the cases the bleeding comes from a spot on the septum about half an inch distant from the anterior margin. The bleeding in these cases may

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usually be stopped at once by introducing a small pledget of iodoform gauze and compressing the nose between the finger and thumb.

I remember the case of an old gentleman who, in a previous attack of epistaxis, had been through all the misery and risk of posterior plugging. The attack from which the patient was suffering when I saw him was very severe. He had been bleeding more or less for three days, and as he was eighty years of age he was not a good subject for such a profuse loss of blood. On examining the patient I found blood oozing from the right nostril, which was blocked with clot. I cleared out the nostril by syringing it with water at a temperature of about 110° F. I then found that the bleeding came from a vessel on the septum at the usual site. I treated this in the manner I have already described, and on removing the plug forty hours later I found that there had been no further hæmorrhage, and the patient made a rapid recovery. I have somewhat wandered from the title of my lecture, but I could not refrain from pointing out how treatment is dependent upon diagnosis in these cases.

I would reiterate the diagnostic value of epistaxis. If it occur in a patient with febrile symptoms which in themselves are rather suggestive of typhoid fever, then it may be regarded as strong corroborative evidence. In middle-aged people the combination of epistaxis and hæmatemesis points to cirrhosis of the liver as the cause of the gastric hæmorrhage; profuse hæmaturia preceded by epistaxis in people above the middle period of life would suggest chronic interstitial nephritis as the common cause of the two conditions. In elderly people the history of epistaxis followed by cerebral symptoms indicates cerebral hæmorrhage as probable.

Dr. Leonard Williams has written to draw attention to the proposed visit of the Balneological and Climatological Society to the northern spas; any letters asking for information should be addressed to him at 8, York Street, Portman Square, W. The arrangements are so contrived that at but little cost, and under very favourable circumstances, medical men can make a tour to Matlock-Bath, Dovedale, Rowsley, Chatsworth, Haddon Hall, and many other interesting places. The party leaves London on April 23rd.

CLINICAL LECTURES

ON

DISEASES OF CHILDREN.

Lecture IX.

THE DYSPEPSIAS OF THE SECOND DENTITION.

Delivered at the London Hospital, November 18th, 1902.

By ROBERT HUTCHISON, M.D.Ed., M.R.C.P.,
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Gentlemen,—In a previous lecture you will remember we considered together the digestive disorders of infancy; and to-day I want to turn to the study of kindred disorders in older children, and to speak about what one may term the dyspepsias incidental to the second dentition. There are probably many reasons which render children at about the period of second dentition particularly liable to suffer from digestive disorders. One of these, no doubt, is the confinement and want of physical exercise, as well as the mental over-strain, which are incidental to school life, which is usually begun at about that period. You will find that many cases of chronic dyspepsia in children are certainly attributable to that cause. In the second place, owing to the rapidity of growth at this time of life, there is a tendency for the large demand for food to outstrip the digestive power, so that the stomach is no longer equal to the demands made upon it by the tissues. This demand is apt to be met by an excessive consumption of carbohydrate foods, and that, in the third place, is one of the main causes of chronic dyspepsia in children of this age. Coming closer to this subject, one finds oneself in a difficulty as regards classification, just as one was in the case of the dyspepsias of infancy. That difficulty arises, again, from the insufficiency of our pathological knowledge. We do not know what the pathology of many of these cases of dyspepsia is, but I think one may conveniently make the assumption that they are all of them essentially catarrhal in their nature, and I shall speak of them under three headings. First there are the acute dyspepsias, or, if you like, acute gastro-intestinal catarrhs; secondly, subacute dyspepsias, or subacute gastro-intestinal catarrhs; and in the third place,

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chronic dyspepsias, due to chronic gastric or intestinal catarrh.

Acute dyspepsia in older children resembles very closely the similar disease which we have already described in the case of infants. It begins suddenly, as its name implies, and the chief symptom is vomiting. The vomiting is accompanied by prostration, and often by a high temperature, and on examination you find that the tongue is thickly furred, but that otherwise there is very little to be made out. These cases usually arise from one of two exciting causes. One of these is the consumption of some irritating article of food. What that food is varies in different instances. The stomachs of some children are peculiarly susceptible to irritation by some articles which are eaten with complete impunity by others. Thus some children have great difficulty in digesting greasy things, and these seem prone in them to produce acute gastro-intestinal catarrh. In other cases the cause may be unripe fruit; but of course there are many different kinds of food which may act as exciting causes of the condition. The other great exciting cause which you have to bear in mind is chill. One has repeatedly seen cases of acute gastro-intestinal catarrh brought on in children at about the period of the second dentition by exposure to cold. Thus I have seen it happen very suddenly after sea-bathing. These cases are apt to begin chiefly with stomach symptoms and to end up with intestinal symptoms; in other words, the catarrhal trouble seems to start in the stomach and to extend downwards through the stomach into the intestinal canal. That is particularly the case in those instances in which the exciting cause is some error of diet. In them the intestinal trouble ends with diarrhœa, and the diarrhœa, by carrying away the irritating material, leads to a spontaneous or natural cure. The difficulty which you will have in dealing with these cases is in being sure that they are not something else. You may have difficulty in realising how severe an attack of acute gastro-intestinal catarrh may be; there may be high temperature, severe prostration, and vomiting, with a furred tongue, and you will, in such a case, be apt to regard the condition as due to something more serious than a mere gastro-intestinal catarrh; for instance, you will think of one of the specific fevers, perhaps of meningitis, or some serious abdominal trouble. I have known a case of that sort in which the abdomen was nearly opened under the impression that the child was suffering from appendicitis.

There is a group of cases in which those attacks recur at intervals, in which a child will go for a few weeks and then have an attack of gastric catarrh, and then go on for another few weeks and have another attack, and the mothers will describe them as "bilious attacks." Yesterday, when I was in the hospital, a child was brought with exactly that complaint.

The treatment of acute gastric catarrh is quite simple. In consequence of the great depression you should put the child to bed and withhold all food for a time, or as long as the vomiting is urgent. During that time you may administer sips of iced water in order to allay the thirst. If there is much vomiting there is no occasion to wash out the stomach, but in cases where nausea is present you may find this lavage of the stomach, as in the case of little babies, an important help. Drugs are not of much assistance to you, but the most useful are calomel and bismuth. You will begin at the outset by giving small repeated doses of calomel, $\frac{1}{6}$ to $\frac{1}{4}$ grain every two or three hours until two grains have been taken; then follow that up by bismuth in large doses. Under that treatment the attack rapidly subsides. And then you have to consider how you are to prevent recurrences of the attacks in future, particularly when you are dealing with a child who is the subject of so-called "bilious attacks." How you are to do that we shall consider when we come to speak of chronic dyspepsia.

We may turn next to *subacute cases of gastro-intestinal catarrh*, a condition which I think is less common than the above, but which may be left behind after the acute attack has passed off, although it may also arise spontaneously and be subacute from the outset. In these cases the fever is not so high as in the acute attacks, but it is more prolonged, lasting perhaps a week or ten days. And as the attacks are accompanied by a tendency to vomit, and sometimes by diarrhœa and a furred tongue, you will readily understand that these cases are very apt to be mistaken for typhoid fever, and many of them undoubtedly used to be written down as "gastric fever." You know that nowadays the term gastric fever has fallen into disuse, and we know that a large

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number of the cases so described were undoubtedly cases of enteric fever, mild in degree. But I think it is also true that some of them were really instances of subacute gastro-intestinal catarrh. Fortunately, in the agglutination test we have a means of distinguishing those cases from true enteric. Clinically, without that test there is difficulty, because the two diseases simulate one another very closely. So you must be on your guard not to mistake them for enteric, and *vice versâ*. The treatment of them is very much the same as that of acute attacks—namely, rest in bed, restricted diet, and the administration of calomel and bismuth.

The last group of dyspepsias is the most important of all—that of *chronic cases of gastro-intestinal catarrh*. These cases are extremely common. You will meet them in some form or another in practice almost every day. Together they form a class which is fairly definite, although one can distinguish in it certain special types of the disease. They are sometimes known under the term "mucous disease," that name having been first given to them, so far as I know, by Dr. Eustace Smith, for reasons which we shall see immediately.

I want to describe to you what those cases of mucous disease are like, and to draw for you a sort of clinical picture of the condition. The child will usually be brought to you by its mother with the complaint that it is wasting. She may tell you in addition that it is languid and peevish, and that it does nothing but "lie about" all day. Very frequently you will hear the complaint that the child has a cough, and the combination of wasting and cough is often the thing chiefly noticed by the mother, and it is apt to throw you off the scent and lead you to believe that you are dealing with a case of pulmonary tuberculosis. Over and over again such cases are sent to hospital with the diagnosis of phthisis. When you come to inquire further into the symptoms of the child you will usually be told that the appetite is very poor. Sometimes, on the other hand, you find quite the opposite condition of things, for there is a voracious appetite. The mother will say that in spite of the fact that the child eats a great deal it does not seem to get any fatter. In those cases, where the appetite is voracious, you will sometimes get a history that the child has a liking for some quite abnormal things. You may find that it has been eating coal and cinders, or has been going to the wall and picking off pieces of plaster and eating them. To that condition of abnormal appetite the term "pica" has been applied, and it is not very uncommonly met with among children who suffer from this special form of chronic gastro-intestinal catarrh. When you inquire into the condition of the bowels you will generally be told that there is constipation. You will hear that the child's motions occur at irregular, and sometimes prolonged, intervals; that the stools consist of more or less hard lumps, surrounded often by material which the mother compares with white jelly,—that is to say, by mucus. Very commonly you will get a history that the child is troubled with worms. Less frequently there will be a tendency to diarrhœa, and one characteristic of such diarrhœa, which you will often notice, is that it tends to come on immediately after the taking of food. The mother will tell you that immediately the child eats a meal there is a tendency for the bowels to act. To that condition the term "lienteric diarrhœa" is applied. Proceeding further in your investigation of the case you will find that these children usually sleep badly at night, that their slumbers are restless and disturbed by unpleasant dreams, and there may even be a tendency to somnambulism. Many of them also have nocturnal incontinence of urine. The mother will frequently tell you also that the child becomes very pale at intervals; she will say that he goes "deathly white" for a few minutes, may be repeatedly throughout the day. These fits of pallor are a little difficult to explain. Some people attribute them to colic. Dr. Angel Money has described them as cases of "vaso-motor epilepsy," because he supposed they were due to spasmodic discharges from the vaso-motor centre analogous to the spasmodic discharges from the brain cortex in epilepsy. I need hardly say that that is a purely theoretical explanation. Certainly they are associated with vascular alterations, perhaps spasm in small arteries or sudden lowering of the blood-pressure; but what the exciting cause of those alterations in blood-pressure is we do not know.

When you come to inspect the child for yourself you will usually find that it is at or about the period of second dentition; the majority of the cases occur in children between the ages of six and eight. The child usually looks poorly nourished; the complexion is pale, and frequently there are

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dark rings under the eyes. On examining the tongue you will find that it presents certain well-marked features. As a rule it is covered by a thin fur, through which you can see projecting red points, which are the enlarged fungiform papillæ. Or, in other cases, and these perhaps the most typical, the tongue has a glazed appearance, and is of a slightly yellowish or fawn-coloured tint, looking, as it has been described, as if it had been brushed over with a solution of gum. In yet a third group you will find the tongue shows peculiar mapped-out areas, curious irregular patches where the epithelium seems to be denuded, which have been fancifully compared to the continents in a map of the world; hence it is called the "geographical tongue." On proceeding to examine the child further you will find that the skin is dry and harsh. Usually you cannot make out any physical signs of disease in any of the great viscera; the heart, lungs, and abdominal organs appear to be healthy. But when you come to inspect the throat you will almost invariably find that it is in a more or less unhealthy condition, that there is chronic pharyngitis, more or less enlargement of the tonsils, and very frequently adenoids in the nasopharynx. It is really to the unhealthy condition of throat that the cough in these cases is due, and that fact I cannot too strongly impress upon you. The majority of cases of cough which are brought before you in children are cases of throat-cough, and not lung-cough at all. When you examine the urine you will often find that it is turbid, owing to a deposit of urates. Sometimes in such a case the mother will describe the urine as being "milky" when passed, that appearance being due to pale urates. On chemical examination you will often find that it contains proteid, probably in many cases nucleo-albumen. Such children make up a large proportion of the so-called "cyclic albuminurias," which are, I think, better termed simply functional albuminuria,—that is to say, cases in which there is no organic disease of the kidney, but in which albumen or proteid appears in the urine at particular times of the day, usually after the child has got out of bed and is going about. The morning urine which has been secreted in the night is usually free from any trace of albumen at all.

Such are the chief symptoms of a typical case of this so-called mucous disease. But you will frequently meet with cases which are not so typical as that, but in which one or other group of symptoms tends to overshadow the rest. And I think you can distinguish several such special groups. First you can distinguish those in which the *constitutional symptoms predominate*. By that I mean that the chief symptom is the wasting, accompanied by mental depression and languor, and perhaps by some degree of mental irritability. Then there is a group in which the *stomach symptoms predominate*. The chief complaint in those cases is that there is pain after food—a pain which you will frequently have difficulty in referring to the stomach or colon, but which may probably have its seat in both, owing to flatulent distension of the stomach and large bowel. Other cases which certainly belong to this great group of intestinal disorder will be brought to you simply for *worms*, and I want to impress upon you at this point that you should always regard worms as a symptom and not as a disease. They are a symptom of chronic intestinal catarrh, and not a disease in themselves. Other cases will be brought to you solely with the complaint of *lienteric diarrhœa*—that is to say, the diarrhœa is so prominent a symptom as to overshadow all the rest. And lastly, you will have cases brought to you for *restless and disturbed nights*, the so-called pavor nocturnus or night terrors.

We will pass on now to glance at the pathology of this disease so far as we know it. It is difficult to speak at all dogmatically in this matter, because there is an almost complete absence of post-mortem evidence in regard to it. It is not a fatal disease; it leads to much ill-health, and it predisposes the child to suffer from more serious disorders, such as tuberculosis, but it is not in itself inimical to life, and thus there is no opportunity for investigating its morbid anatomy. Dr. Eustace Smith has a very neat theory to explain all the symptoms detailed above. He says that mucous disease is due to an over-secretion of mucus throughout the whole alimentary canal; that the excess of mucus produces in the stomach sluggish and painful digestion; in the intestine it wraps round the intestinal contents, the consequence of which is that the digestive juices get imperfect access to the food, and hence absorption is interfered with, and as a result the child wastes. Moreover the scybalous masses in the large intestine get coated

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over with mucus, so that the wall of the gut gets no purchase over them to squeeze them along, but glides over the masses; hence the frequency of constipation. Moreover the mucus forms a good nidus for the worms to live and feed and lay eggs in. One might go further and say that in the cases in which there is proteid in the urine it is very often a nucleo-proteid, and that that is due to analogous over-secretion from the cells lining the urinary passages. And one might also point to the throat and say that there, too, one finds chronic catarrh and the production of an excessive quantity of mucus. This theory, then, as you will see, is very neat and ingenious, and it does explain fairly satisfactorily most at least of the symptoms. But it is one which it is very difficult to be sure about. In those cases in which you get the passage of masses of mucus from the bowel there is a considerable amount of evidence in support of it. But this does not occur in by any means every case, and the real importance of the theory is that the treatment which is based upon it is certainly justified by its results. That treatment I shall describe to you immediately. There are many who do not accept the mucus theory, but who consider that these cases are more obscure than any such explanation as that would lead you to believe. They think one is here dealing with a disorder of metabolism, a defective power of assimilation which they compare to the gouty condition in grown-up people. They say—and especially French authors—that those children have the "arthritic diathesis," whatever exactly that may mean. There are people in this country who also maintain that view and believe that these cases are due in some way not completely understood to an imperfect elaboration of proteid material, which leads to the production of an excess of uric acid, pretty much as you know takes place in gout. Between these two theories I shall not attempt to decide, because, as I have said, there is no definite ground to go upon in the argument. I want further to point out to you that whatever the true theory of the mode of production of the symptoms is, there can be no doubt whatever about one frequent exciting cause of the disorder, and that is a matter which it is of more importance for you to know. It is the excessive consumption of sugar. That is so common and so well recognised a cause of this disease that you might almost call it the dyspepsia of sweet-eating children. You will remember that cane sugar is eminently a producer of mucous secretion. If you take cane sugar into the mouth and suck it the mouth becomes sticky, not merely from the presence of the sugar, but from the secretion of a viscid mucus by the salivary glands. So that this fact would also fit in quite well with Dr. Eustace Smith's theory.

We come now to the diagnosis of chronic gastro-intestinal catarrh. I have said that these cases will come before you with great frequency, and there is one condition which you are particularly apt to mistake them for, and that is tuberculosis in some form. One reason for this is, that the child is so often brought for cough and wasting, a combination of symptoms which in a grown-up person you are apt to associate with the presence of pulmonary tuberculosis. But I would ask you to remember once and for all that pulmonary phthisis is a disease almost unknown in children of this period of life. Tuberculosis at this age does not tend to take the pulmonary form. If you are in doubt whether you are dealing with a case of latent tuberculosis somewhere, I advise you to give heed to the temperature; make a point of taking the temperature night and morning for a few days. Dyspeptic cases are not accompanied by fever, or, at all events, the fever is not continuous. There may be transitory attacks of fever during the exacerbations of the condition, but these only last a short time, whereas cases of tuberculosis, no matter where situated, tend to be accompanied by nocturnal rises of temperature, which persist for several weeks.

Finally we arrive at treatment. If you set about this in the right way I think I can promise you very satisfactory results. But if you are not familiar with these cases you will find them very troublesome and chronic and difficult to cure. Seeing that I have just mentioned that the excessive consumption of carbohydrate food, particularly sugar, is undoubtedly one of the chief exciting causes of this form of dyspepsia, the main line of treatment must be dietetic. The first thing to do is to attend to the food. The sheet I have put into your hands describes to you the chief lines upon which the diet for such cases should proceed.

Directions for Diet in Chronic Dyspepsia.

No food must be taken between the regular meals.
No sugar or sweets of any sort are to be eaten.

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The child must *not* eat :—New bread, potatoes, peas or beans, turnips or carrots, pickles, pastry, jam, syrup or treacle, or cakes.

The diet should chiefly consist of :—Stale bread or dry toast, with butter or dripping, bacon, eggs, fish, meat, milk, plain milk pudding (except cornflour, arrowroot, or sago), and green vegetables in small quantity.

You will see that it consists essentially in the limitation of carbo-hydrates, and in the almost total prohibition of sugar. If you will keep that broad principle before your minds you will have no difficulty in adjusting the details. Next to a change of diet I put in order of importance change of air. Repeatedly one has seen cases which were hanging fire and not doing well get better right away when they were sent to a convalescent home at the sea-side. That is probably to be explained on the grounds which I have already mentioned, that many of these cases are due to a lack of physical exercise and confinement to school for long hours, and to unhealthy homes. Then we come to consider the question of drugs. Of what help are drugs likely to be in the treatment of these cases? You will find that the drugs chiefly of use at the outset of the case are alkalies and aperients. I think aperients are useful no matter what the state of the bowels—whether there is constipation or not,—and it is a good routine plan to begin by using them. The usefulness of those remedies may also be accounted for on the excessive-production-of-mucus theory. According to the holders of that theory you administer alkalies because they are an admirable solvent of mucus. And you administer aperients so as to get rid of the mucus which has been so loosened. Whether the explanation is right or not, at all events you will get great success from the adoption of this line of treatment. I use some such prescription as this :—Pot. Bicarb., Pot. Cit., āā gr. v, Tinct. Nucis Vom. ɱj, Compound Infusion of Gentian ʒij, given with a little water a quarter of an hour before the meals. The action of each ingredient of this prescription is believed to be this. The bicarbonate of potash dissolves the mucus. The citrate is given largely because these children tend so often to have thick urine and the passage of urates. That, acting as a diuretic, tends to improve the condition of the urine. The nux vomica is given to increase appetite, which is usually bad. The gentian is given for the same reason, and also because it has a mild astringent action on the mucous membrane of the stomach. The aperients I like to begin with consist of rhubarb and mercury in the following combination :—Rhubarb powder, 8 grains ; bicarbonate of soda, 10 grains ; grey powder, 1 to 2 grains, according to the child's age. This should be given every night, or in a milder case every other night. Rhubarb is selected because it has not only an aperient action, but it has an after astringent action, and a tonic effect upon the flabby mucous membrane. One adds bicarbonate of soda for the same reason as in the other mixture ; and many cases are undoubtedly greatly benefited by mercury in some form or another, probably because the mercury seems to have a peculiar specific influence in diminishing catarrh of the alimentary canal. After a course of such treatment for a fortnight or so you will probably find a striking degree of improvement ; the child's appetite will have returned ; it will begin to gain flesh. It will no longer suffer from restless nights, and you will have reached a stage at which you can begin the administration of tonics, which must include iron in some form or other. A very good form is the wine of iron, given with an aperient in the form of aloes. I want particularly to impress upon you that you should defer the administration of those tonics, particularly iron, until you have got the tongue clean and the stomach in a healthy condition. There is no commoner mistake than to treat these cases at once with cod-liver oil and Parrish's food as soon as they are seen. The doctor is apt to order them iron to make them strong and cod-liver oil to make them fat ; and such treatment, if begun too early, does harm instead of good. It is a good rule in medicine not to begin the administration of iron until the stomach is in a healthy condition, because iron is almost always an irritant to the stomach, and it is not absorbed and made use of until the alimentary canal is brought into a condition somewhat approaching that of health. Although that should be your general line of treatment, you will find that special symptoms in particular cases may call for special remedies. Many patients, for instance, are troubled with gastric pain or colic, which may be extremely obstinate, and sometimes the only thing to which it will yield is opium. The best form in which to give it is Dover s powder, and two or three grains ought to be sufficient. If you have

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lienteric diarrhœa to deal with, you will find the best thing to do is to give small doses of opium before meals—perhaps ɱij of the tincture of opium may be enough,—and it is well to combine with that a drop of Fowler's solution and a little nux vomica. Lienteric diarrhœa is brought about by peristalsis in the upper part of the bowel being too active and too easily excited reflexly, so that when food is introduced into the stomach, instead of only exciting the stomach it excites a wave of peristalsis, which spreads through the intestine and leads to a motion. Opium is often the only thing which will allay that and prevent the diarrhœa. The existence of worms may be a prominent feature, and, as I have said, you should never regard these as a disease, but as a symptom of an unhealthy condition of the mucous membrane, which makes the lodgment of worms possible; and so in these cases, more than in any other, you should administer purgatives to clear away the mucus, and in very bad cases you can combine that, if you like, with enemata of salt solution or quassia, both of which you know act as parasiticides. It is only very rarely that you will find it necessary to resort to santonin. I almost never prescribe it. I usually find that regulation of the diet and the administration of aperients get rid of the worms and put the mucous membrane of the bowel into a healthy condition, so that their further lodgment in it becomes impossible. Should headache be a prominent symptom, as it is apt to be, I recommend you always to pay particular attention to the refraction of the eyes. You will often find that there is a slight error of refraction, astigmatism, or hypermetropia, and if you correct that by means of suitable spectacles you often find that the headache disappears at once. The cough, which is so common in these children, may necessitate local treatment directed to the throat; you may require to use nasal douches, or washing out of the back of the throat with an alkaline and antiseptic solution. But in many instances you have to proceed sooner or later to the removal of the tonsils, and perhaps also of adenoids. But the relation of adenoids to these cases I shall discuss more fully when I come to speak of their medical bearings in children in general.

WITH DR. JAMES GALLOWAY AT THE POLYCLINIC.

(*Continued from p.* 352.)

Late Cutaneous Syphilide.

This next patient came to me to-day, having been sent across by my colleague, Dr. Montague Murray. The case is not severe, but it gives opportunity for the exercise of some skill in diagnosis, so I asked her to come along and show herself to you. The history, as far as I have been able to ascertain it, I will tell you after you have seen her. I will now simply ask you to look at the case and form your diagnosis. Now the patient has gone I may say she shows a very well-marked and characteristic example of a late syphilide, and the history is that she has been in very good health except that she has suffered slightly from chronic rheumatoid arthritis. She has been married for many years. Her youngest child is nineteen years of age. The history of her succession of children is—the first child born seems to have been healthy, but has been to the hospital suffering from what appears to be dyspepsia; she is married. Then there were two miscarriages, and then there were three healthy children. But I do not think it is necessary for our diagnosis to inquire minutely into the history of a case such as this; we should test if we are sufficiently well trained to recognise such a condition, for we can judge of its nature from its physical characters without any further inquiry. This eruption on the forehead commenced three years ago only. First there was a little pimple on the centre of the forehead,—that is to say, a small nodule, for it did not suppurate. This grew and spread peripherally, or, as the patient herself said, it always spread like a half-circle. You will notice the very characteristic symmetrically circinate distribution which it has at the present time. There is a similar lesion between the thumb and the forefinger of the right hand. I want you to notice this specially, as the condition should not be mistaken for chronic eczema of the palms. If you pinch that up you will find that the skin wrinkles quite differently from the normal skin, and it does so because the cutis is infiltrated. The infiltration of the lesion on the forehead and the infiltration of the lesion on the hand—infiltration in the true sense, that is to say, a pouring in

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of the newly formed cells,—the peculiar rounded cells of syphilis—produce the characteristic feature of a syphilitic eruption. It is this infiltration which is of importance in the diagnosis between the eruptions of a syphilitic type and those in certain other diseases for which they may be mistaken. There is no reason to mistake this for psoriasis or lichen planus, or the erythematous seborrhœic dermatitis. In psoriasis and seborrhœic dermatitis you are dealing with a disease in which the visible changes occur mainly in the epithelium. In lichen planus there is an infiltration in the true skin, but the nature of it is different. Then there still remains the diagnosis between this disease and cutaneous tuberculosis, which is the most important alternative to distinguish it from. One point alone in this case is sufficient, without going into anatomical considerations, to guard you from making that diagnosis. The fact that it commenced three years ago, and that it has affected so much of the skin already, is sufficient to make the diagnosis positive. The infiltration does not distinguish it readily from certain other forms of specific granuloma, but its duration distinguishes it from the condition which is most likely to be confused with it, namely, very superficial cutaneous tuberculosis. The trouble in the treatment of many cases of facial syphilis is not so much in curing the specific disease, but, when the eruptions have been allowed to ulcerate usually as the result of mixed pyogenic infections, in obtaining a good scar. A scar forms, resembling somewhat the scar of cutaneous tuberculosis, and you know how difficult it is to get the scar of lupus which has ulcerated to have a healthy appearance. After you think the lesion has healed it breaks down and ulcerates again, although there may be no recurrence of the tuberculosis. At the present time I do not propose to give this patient any mercury until we see how much of the granuloma will be absorbed by the action of the iodide of potassium. Later on I think we may give her mercury to rid her, if possible, of her syphilitic infection.

Diagnosis of Superficial Cutaneous Tuberculosis.

Our next patient is this man. For purposes of comparison with the last case I wish he had presented one of the other varieties of cutaneous tuberculosis, resembling syphilis still more closely. He says he developed the disease when he was twelve years of age,—that is to say, twenty years ago. It commenced as a papule in the centre of the cheek, and gradually spread, involving the surface widely. It was scraped first of all when he was thirteen years of age, and again when he was about seventeen years of age. Then it seemed to have remained quiescent for a considerable time, and he pursued his work as a builder and plumber at Eastbourne. Three years ago the disease commenced to spread a good deal more rapidly in the nasal and orbital directions, and now he comes up for treatment. I draw your attention to the fact that we have here an example of one of the rarer forms of tuberculosis, which is not infrequently mistaken for lupus erythematosus. The time at which it commenced ought to give you some indication of the nature of the disease; lupus erythematosus is comparatively rare in young people, but that is not the criterion which will distinguish between lupus erythematosus and this condition. Here, as in our last patient, especially in the portion which has been spreading recently, the characteristic infiltration of the granuloma is seen. There is something in the skin; it is not an epithelial disease like psoriasis; here there is an actual infiltration, as if some wax-like material had been injected into the cutis; that is the important point to bear in mind in the diagnosis of such rather unusual forms as this, and the cases which you have seen of syphilis. You notice a little portion of it has become verrucose, a condition not uncommon in chronic forms of cutaneous tuberculosis. I think we shall do well in the treatment of this case. He has promised he will remain continuously under treatment for four months, and subsequently twice a week if necessary, and continuity of treatment is of the utmost necessity. A rapid plan, and very satisfactory one, is by "multiple scarification;" and if that is done carefully it is wonderful how little scar is left, and how very good the scar can be. But these cases also react very well to Finsen's light treatment, and though the method is a very slow one, the scar which is ultimately produced is excellent. I feel inclined to attack, at any rate, the nasal and orbital portions by means of ultra-violet light rays, and perhaps combine with that some other form of treatment, of which multiple scarification would perhaps be the most satisfactory.

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A POST-GRADUATE LECTURE

ON

WHITLOW AND CELLULITIS.

Given at the West London Hospital, January 13th, 1903.

By STEPHEN PAGET, F.R.C.S.,

Surgeon to the Hospital, and Surgeon to the Throat and Ear Department of the Middlesex Hospital.

DURING the last few months a great number of septic cases, of a very severe type, have been admitted under my care here—sufficient, I think, to justify the belief that cases of this kind are especially frequent during the late autumn and early winter. Among them, there have been cases of pyæmia, acute gangrene, and carbuncle of the lip; more than one case of acute periostitis; and many cases of cellulitis, which began, most of them, as ordinary cases of whitlow. In one of these cases of whitlow and cellulitis, I was compelled to amputate the forearm; and I have lately heard of a similar case, in another hospital, which ended in gangrene, amputation at the shoulder-joint, and death a few hours after the operation. So I do not apologise for taking such a familiar subject as a whitlow; because these two cases, which ended one of them in death and the other in mutilation for life, are evidence that we cannot tell, of a whitlow, what it will come to.

And I should like to say first of all, concerning whitlow and cellulitis, that many patients come here who have already received treatment at home, and that the treatment has been, in many instances, wholly inadequate. We get cases where the finger is all infiltrated with pus, and there is pus in the palm, and pus burrowing among the tendons at the wrist; and somewhere near the tip of the finger there is a little cut, less than an inch long, made without an anæsthetic, and of no use at all. It is the same with some of our cases of cellulitis; they come here in urgent need of four or five times as much incision as they have received. It is almost impossible, in a bad case of whitlow, to do without

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an anæsthetic. In some cases it will suffice to freeze the skin with ethyl chloride; but, wherever it is practicable, a general anæsthetic should be given. It is true that cases do occur which can be cured by a very small superficial incision; but these are the exception, not the rule. Indeed, with a finger, there is no distinction between superficial structures and deep structures. I think that we are too ready to consider a finger as though it were a much larger and more complex thing than it is. After all, a finger is only a short terminal process or extension of the tissues of the hand. The bones and tendons have no chance of escape from infection; for what is a finger, except skin and bone, and some tendons lying between the skin and bone? When a finger is acutely inflamed, every one of its tissues, within the area of inflammation, is affected; there is no distinction, no line of demarcation, between skin and bone and tendons; they all suffer together, and they all suffer alike, except that bones and tendons are tougher than skin. Therefore, if the flexor aspect of the finger needs incision, the knife must be carried well down to the tendons; if the sides of the finger need incision, it must go well down to the bone. It is better to cut a finger almost to ribands than to let the tendons slough in their sheath, and so infect the whole hand.

Whatever we do, some of these cases of whitlow will come to amputation of the finger, sooner or later. Some of them come to it sooner; that is to say, the patient arrives here with a finger that is past saving, the skin undermined and rotten, with one or more holes in it, the flexor tendons strangulated and bulging out of their sheath, and the bones here and there grating under the probe. In such a case a day or two may be spent in cleaning-up the finger with baths and fomentations, and in improving the patient's health by food and wine, and tonics by day, and narcotics at night; and then, the sooner the finger is off the better. I think it is best, in such cases, not to remove the head of the metacarpal bone, and not to expect primary union; but to put in a few points of suture, and to leave the rest of the healing to be done by granulation-tissue.

In other cases, these fingers come to amputation later; that is to say, by free incision and careful and tedious dressing and splinting, the finger is "saved," and the surgeon rightly congratulates himself on his skill; but, a few weeks or months later, the patient comes back with the finger more or less flexed, stiff, wasted, cold, and painful; it is always in the way, it catches in everything, and is a constant worry to him. In almost all cases of this kind it is best to remove the finger, and have done with it. Of course, no two cases are alike; it may be right, in this or that case, to try to make the finger straight and flexible and useful again by open division or splitting of webs and bands, and by skin-grafting. This was done with success in one case, a few weeks ago, by Mr. Pollock, my house surgeon at that time; but this operation is not always practicable. As for the forcible movement of these withered fingers under an anæsthetic, I believe that it is practically no good at all; and, in nine cases out of ten, the patient and the surgeon will agree that the finger is better off than on. But, of course, the head of the metacarpal bone should be left. Indeed, I cannot see what right we ever have to remove this useful and innocent bit of bone; the loss of it does not really improve the look of the hand, or conceal the absence of the finger, but it does impair the grip and the dexterity of the other fingers.

There is another point to be considered: whether the cut ends of the flexor tendons ought to be stitched in the wound or allowed to slip back into the palm of the hand. It is said that if they slip back they may carry infection with them. I do not think there is much meaning in this phrase; if the cut tendon is so unhealthy as that, it will not be disinfected by any amount of stitching. Practically, I have never seen any harm come of leaving the cut tendons to look after themselves; and, if they do retire up into the palm, they leave the more room for the natural drainage of the tendon-sheath.

With regard to pus in the palm of the hand, I venture to say that we need not be afraid of hæmorrhage when we incise the palm. Of course we are bound to keep in front of the line of the superficial palmar arch, though nothing very bad would happen even if that line were transgressed. Anyhow, I cannot recall any case of severe primary hæmorrhage from incision of the palm. A case of secondary hæmorrhage, which I do remember, was cured by ligature higher up: but I do not remember ever seeing difficulties from bleeding at the time of incision.

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So also with cellulitis: I have never seen any difficulty or danger from primary hæmorrhage in any incision made for the relief of cellulitis, and I can recall only one case of secondary hæmorrhage; but this was a case of so great interest that I should like to say something about it. The patient was a boy, in whose hand and forearm several incisions had been made; secondary hæmorrhage occurred through an incision a little above the wrist. Plugging stopped it for a time, but it recurred again and again; Mr. Seymour, my house surgeon, therefore tied the radial and ulnar arteries at the wrist. Then a few days later there was again a sharp hæmorrhage, and it became evident that it must be coming from the anterior interosseous artery; so Mr. Seymour then tied the brachial, and after that the boy made a good recovery without any further trouble. Of course, before the brachial was tied many attempts were made, by careful pressure, and by opening up the bleeding wound, to stop the recurrence of the hæmorrhage; but they failed, and Mr. Seymour wisely tied the brachial.

The word "cellulitis" has not a very exact significance: it is applied alike to cases where there is pus, and to cases where there is no pus; to cases with an external wound, and to cases without an external wound; to cases that require incision, and to cases that will get well without incision. In hospital practice nine cases out of ten do require incision; we have to deal with an acute diffuse inflammation of the skin and subcutaneous tissue, tending to cause death and sloughing of the deep fascia. Now and again it may be well to wait ten or twelve hours, to give the inflammation a chance of subsiding under perfect rest and careful fomentation; but in most cases it is mere waste of valuable time; and in a bad case, with marked fever and constitutional disturbance, and the limb greatly and uniformly swollen, bright or dusky red, hard, tender, and painful, with soft spots here or there which are even more sensitive than the rest of the limb—in such a case it is worse than useless to wait. As for the number and length of the incisions, it would perhaps be true to say, in a bad case, that they ought to be about three inches long and three inches apart, and ought to go their whole length right down to the deep fascia. Then comes the question—ought we to go through the deep fascia, with a knife or a director or a sinus-forceps, to try to find pus burrowing among the muscles? In ordinary cases of cellulitis I am sure that we ought not to do anything of the kind. Cellulitis may, indeed, co-exist with pus burrowing from the palm, under the annular ligament, and up among the flexor tendons above the wrist, and this pus may need to be let out, by making a way down to it; but that is no reason why we should go poking among healthy muscles at the risk of carrying infection into them.

With regard to the treatment after incision, I think it is best to lay gauze lightly in the wounds, and to wrap the limb in gauze for a few hours only, and then to foment it with hot boracic lint. It is a mistake to attribute any great value either to drainage-tubes or to plugs of gauze. I have seen drainage-tubes kept in day after day, even after they had made regular tunnels that you could look through and see daylight at the other end; and thick, sodden plugs of gauze pushed in at one incision and out at another, and moved and pulled about, giving pain at every dressing, and serving only to keep the pus and the sloughs from finding their way out. I am sure that this excessive use of tubes and plugs is bad surgery. And the same applies to the excessive use of the arm-bath. It is very tiring and uncomfortable to keep one's arm in a bath, and I doubt whether a bath for an hour is any better than a bath for a quarter of an hour. Nor is there any evidence that it makes any difference what sort of antiseptic you put in the bath. Probably a dash of iodine or of Condy's fluid is just as good as anything else; and a hot soaking for ten or fifteen minutes is quite long enough. Then take the arm out, wrap it in hot fomentations, and leave it alone for a time. Like every wounded limb, it wants rest; and it cannot get properly rested if it is perpetually being bathed and plugged and moved about.

As for the constitutional treatment of cellulitis, I should like to express my disbelief in any preparation of iron in erysipelas, cellulitis, or any similar septic state, and my firm belief in quinine in five-grain doses four times a day. These cases generally take quinine well; in one case, a boy with pyæmia, who finally recovered, I gave the very large quantity of eighty grains a day for ten days. And I do firmly believe, also, in the use of antistreptococcic serum; provided, of course, that the infection has been proved to be the work of streptococcus, and not staphylococcus. In cellu-

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litis, and in operation-wounds and dissection-wounds, and in puerperal fever, there is plenty of evidence that the serum is of great value, only it must be used early; and a lot of fault has been found with it which ought to be attributed not to the serum, but to the delay till many days, or even many weeks, after infection has occurred.

I should like to say one thing more about drugs. We sometimes want to prescribe a really good tonic which shall contain neither quinine nor iron, nor quinine and iron. Let me give you one of Sir James Paget's favourite prescriptions; I have written it at his dictation, I know not how many times, when I was his secretary. Six drachms of the compound tincture of cinchona in eight ounces of the decoction of red cinchona; an eighth part twice a day before meals in not less than a claret-glassful of water. And he used always to add this advice, that the prescription must be made up by a good chemist, so as to be sure that the decoction was freshly made. In these days of tabloids it may be worth while to bear in mind a good old-fashioned mixture, which was administered with marked success to a number of very admirable people in the Victorian Age.

And I should like to say a word about the value of sleep in these cases. Most of them when they come here have already had a week or ten days of pain, with broken nights, and they are feverish and miserable. Free incision will doubtless improve their general condition, but it will not at once stop their pain; what they want is two or three good nights' rest. In all such cases it is best, I think, unless there is some clear reason against it, to give a hypodermic of morphia and strychnine. There is no antagonism between these two drugs thus given, and I have never known anything but good come of administering them together.

As for the feeding of these cases of cellulitis, and all cases of a similar kind, I venture to say that it is almost impossible to over-feed them; I like to see milk and strong beef-tea, and beaten-up eggs, and wine or brandy, simply poured into them. Not long ago a young man was admitted into Accident Ward with acute periostitis of the whole length of the shaft of the tibia; and though Mr. McKay made immediate and very free incisions, he soon had pus in the ankle-joint, and septic pneumonia. He was in a typhoidal state, half unconscious, and losing flesh with most amazing rapidity; and I found that he was having only four ounces of fluid food every two hours. If he had gone on with this absurdly small quantity of nourishment, he would certainly not be in Accident Ward now. We must pour food into such patients by the pint: and if they are delirious and will not swallow it, we must immediately tie them down and feed them through a soft nasal tube.

The worst cases of cellulitis, those who are most in danger of dying, and are slowest getting well, are not the big, full-blooded, alcoholic men, but the feeble, over-worked, half-starved women; they get into a chronic septic state, which may last for weeks; they do not pick up, they do not throw off the disease. We want those old words back again, the *sthenic* type of disease, and the *asthenic* type. We have kept the latter word, as a part of the word neurasthenic; but we want it by itself; the cases that I am thinking of are in no sense neurasthenic, but they are asthenic, and they need very generous feeding, and very careful and constant nursing.

Finally, let me say something about that most terrible and fatal form of cellulitis, the so-called carbuncle of the lip. Of this I have had three cases; two occurred some years ago, and one a few months ago. Two died; and the fact that one recovered is, I think, almost evidence that it was not a genuine case of the disease. Perhaps we may take this definition of the disease—that it is an acute diffuse inflammation of the face and neck, beginning round a pustule on or near the lip, and going on very rapidly to purulent infiltration of the tissues, with septic thrombosis of the veins, septic absorption, and death. The pustule, which is the starting-point of the infection, looks to the naked eye just like an ordinary boil; and the infecting organism, under the microscope, looks like an ordinary staphylococcus; and, though it is easy to invent explanations, we do not know why the disease should run this frightfully virulent and rapidly fatal course. We may call it angina Ludovici, but we gain nothing by giving it a Latin name; or we may attribute the intensity of the infection to the looseness and vascularity of the tissues infected; but this, I think, is mostly guess-work. Anyhow, these terrible cases do occur. They begin as a common-looking boil near the lip, such as one might put down to shaving with a blunt razor; it is neglected, or made worse by

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picking and scratching; then the cellulitis begins, and the patient probably poultices it; and then, a day or two later, he comes here. At first sight the gravity of his case may be overlooked; but in two or three days' time the facial veins will be filled with septic thrombi, and the whole lower part of the face, and the front of the neck, will be infiltrated with septic lymph. You may cut his lip to ribands then, or you may slash his face regardless of scarring, and still he will die; the tissues seem unable to drain, unable to suppurate healthily; it is not like cutting into an abscess-cavity, it is like cutting *post mortem* into a lung crammed full of tubercle; nothing comes out, nothing flows away; you only cut everywhere into firm, brawny tissue, riddled with pellets and plugs of semi-solid purulent lymph; the patient passes rapidly into a profoundly typhoidal state, and dies of acute septicæmia before free suppuration can be established. There is no treatment but incisions; and these must be made very freely, and again and again, and must be carried even beyond the area of infiltration; and, by every possible means, the patient must be kept going, in the faint hope that he may pull through the stage of acute poisoning, and may live to suppurate freely.

SARCINELLI ('La Riforma Medica,' December, 1902) has found the usual methods of percussion of the spleen in infants open to objections. He has devised a method of his own which he describes as follows:—The little patient is held up by the assistant in the horizontal position half-way between the dorsal and left lateral decubitus, one arm of the assistant being passed under the left shoulder and the other beneath the pelvis. The spleen is then percussed by the operator from below. By this method the spleen, which is easily displaced by reason of the lax ligaments which support it, falls by gravity to the posterior surface of the costo-abdominal parietes, there presenting a larger area for percussion than in any other position; and being carried away from the stomach, this, if distended with gas, does not mask the percussion-note of an enlarged spleen.—*Philadelphia Med. Journ.*, March 7th, 1903.

TWO LECTURES
ON
DIABETES MELLITUS.

Delivered at the Post-graduate College, West London Hospital.

By A. E. RUSSELL, M.D., B.S.Lond., M.R.C.P.,
Assistant Physician to the West London Hospital;
Demonstrator of Physiology, St. Thomas's Hospital.

LECTURE I.

A COMPLETE understanding of the pathology of diabetes would presuppose a complete knowledge of the life-history of carbohydrates in the body. Unfortunately such knowledge is at present very incomplete, but none the less a few words must be said about the main theories which are held.

To begin with, carbohydrate food is converted by the agency of the ferments of the saliva and pancreatic juice into a more diffusible sugar known as maltose. This maltose is further acted on by the succus entericus, and by this action and during its passage through the intestinal mucous membrane disappears as maltose and undergoes conversion into dextrose. Dextrose is the form in which sugar exists in the blood, where it is found in health in amounts varying between 0·15 and 0·2 per cent. As soon as the amount exceeds the latter, sugar is eliminated by the kidneys and appears in the urine. As regards the changes undergone by the sugar after passing through the intestinal wall, the old view of Claud Bernard was that it was taken to the liver and there stored up as glycogen; that this occurs is undoubted, and during digestion the portal blood contains much more sugar than the hepatic blood. Bernard further assumed that in the interval between meals the glycogen was converted by a ferment formed in the liver into sugar and passed into the blood for transference to the tissues; his evidence being founded upon analyses of the portal and hepatic vein blood, the latter containing more sugar than the former.

Pavy,* however, as the result of long and able researches into the subject, denies that this occurs. As the result of his analyses he states that the blood issuing from the liver between meals does not contain more sugar than the portal blood, and that therefore the liver cannot be regarded as a

* ee 'The Physiology of the Carbohydrates,' 1894.

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sugar-forming organ. That the liver cannot be the sole organ controlling the sugar supply to the tissues is certain, for at the most it can only contain 150 grammes of glycogen, and a meal rich in carbohydrate food would contain far more sugar than the liver could store, even supposing it to contain none before the meal, a condition which never occurs in health.

Professor Schäfer, in his article on metabolism,* states: "Even, however, if we take into consideration the whole of the glycogen in the liver, that in the muscles, and that in other tissues of the body in which it might be stored, it will still be found that the whole of the carbohydrates of a meal which contains much of these substances is not represented in the body, either by the glycogen of the organs or by the sugar present in the circulating fluid. The amount which is not accounted for may possibly pass into the constitution of the proteids and nucleo-proteids, and also of those albuminoids from which a carbohydrate material has been obtained on decomposition with acids, and it may be that the excess is in this way stored until required."

Pavy cites the case of an animal, such as the rabbit, whose stomach is always full, and whose portal blood always contains more sugar than is found in the blood issuing from the liver; in such an animal, therefore, the liver obviously acts as a check to the passage of the sugar.

Pavy's contention, in brief, is that sugar after absorption is converted by the agency of the cells of the intestinal villi into fat and also built up into proteid, and that the liver is merely an organ which, from the sugar point of view, prevents its passage through into the general circulation, converting it in all probability into fat and proteid. That carbohydrate can be converted into fat may be regarded as definitely settled, and we will not enter into the evidence of such transformation.

The relationship of carbohydrates to proteids is a matter of the first importance, and Pavy has established the view originally enunciated by Schutzenberger that a sugar-reducing substance can be split off from proteids, *i. e.* that all common proteids (casein excepted) have a glucoside constitution.

The fact that a large amount of sugar can be excreted in severe forms of diabetes when no sugar is being administered in the food, concurrently with the increased excretion of nitrogen in the urine, suggests strongly that the body-proteids are being broken down with the cleavage of carbohydrate.

It may therefore be considered as extremely possible that in the passage through the intestinal wall dextrose attaches itself to peptone to form blood-proteid, and that this proteid in its passage through the tissues yields up to them its carbohydrate portion for the purpose of oxidation, the remaining part of the complex molecule undergoing further changes in the ordinary metabolic processes. It is also quite certain that fat can be produced directly from sugar, and also that the liver can store up yet another portion of ingested carbohydrate as glycogen, this portion being, according to Pavy, later converted into fat or proteid.

Looking at the life-history of sugar in the body in this light, it would appear that none ought to be found free in the blood; but the presence of a fairly constant amount of sugar in the blood seems to be undoubted. However, it must be remembered that the blood is the means by which the different foodstuffs are conveyed to their destination. Any fat that it contains could not yield sugar on analysis, so that the origin of the blood-sugar must be sought in that constituent of the blood which may convey the sugar to the tissues, and that constituent is, as already mentioned, to be looked for in the blood-proteids, which from their glucoside constitution must, as Pavy has shown, contain a carbohydrate moiety. Possibly the means which are adopted of analysing the blood may split off the sugar from the complex proteid molecule, and so convey the impression that it is free in the blood. It has also to be remembered that the blood-corpuscles, apart from the blood-plasma, comprise a tissue of considerable size, and that the metabolism of such a tissue must be considerable, and involve the consumption, like other tissues, of sugar among the other foodstuffs; so that one would expect to find sugar in the blood just as it is found in other organs.

The presence of free sugar in the blood is inexplicable on Pavy's hypothesis that the liver is an organ which prevents the passage of sugar into the general circulation.

* 'Text-book of Physiology,' vol. i, p. 917.

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The metabolism of sugar is, then, very complicated, and far from fully understood. Entering as it does into the life-history of fat, of glycogen, and of proteid; intimately connected as it is with the functions of the liver; and a necessary food material to the tissues—it would be surprising if no influence existed which could exercise some control over its changes and its utilisation. The presence of such a factor first became evident in the observations of Minkowski of the production of diabetes after experimental extirpation of the dog's pancreas. This fact is too well known for it to be necessary to enter into any details concerning it, but it may be said that it is settled that it is not the ordinary secreting portion of the gland that has anything to do with the disease, as the common duct may be ligatured or obstructed by disease without necessarily causing any glycosuria. There remains, therefore, that portion of the pancreas discovered by Langerhans in 1869, and since named after him as the cell-islets of Langerhans. These cell-islets are to be found between the acini, and are composed of small, irregular, polygonal cells, with round nuclei and homogeneous refractive cell-bodies. They are not supplied with any ducts, and are surrounded by rich capillary networks.

Now it appears that so long as disease of the pancreas is confined to the ordinary secreting portion no symptoms of diabetes appear, but that when the cell-islets are involved diabetes is present. It is necessary, therefore, to ascertain first in what proportion of cases of diabetes the pancreas has been found abnormal, and secondly, what is the nature of the pancreatic lesion.

Osler* states that in fifteen cases under his notice the pancreas was found on gross examination to be atrophic in nine.

Williamson† thoroughly examined the pancreas in twenty-three consecutive cases of diabetes and found disease in eleven.

The first author to definitely correlate the presence of diabetes with changes in the islands of Langerhans was Ssobalew‡, who reported two cases of diabetes with chronic interstitial pancreatitis in which, on microscopical examination, he could find no traces of the cell-islets. He further found that after feeding dogs with carbohydrates the cells of the islets became granular in appearance.

Shortly afterwards Opie* gave the results of the examination of eleven cases of diabetes, in four of which he found marked changes in the pancreas.

In one the cell-islets were in a state of advanced hyaline degeneration, while a considerable part of the secreting tissue was in a fair state of preservation, though the seat of fatty degeneration.

In one, following obstruction of the ducts by calculi, chronic interlobular pancreatitis was found. In this type the islets are only implicated secondarily when the lesion is in an advanced stage; in fact, they may appear fairly normal, though imbedded in dense scar-like tissue. In this case the diabetes was of mild type and yielded to dieting.

In two, interacinous pancreatitis was found; in a third case of similar affection of the pancreas hæmochromatosis was present, a condition which at a later stage is usually associated with diabetes.

Opie describes two main forms of chronic pancreatitis:

1. Interlobular pancreatitis, with proliferation of fibrous tissue between the lobules which are invaded from the periphery, the islets only being invaded when the process has reached a very advanced stage; the type of such change occurs as a chronic inflammation following occlusion of the duct.

2. Interacinous pancreatitis; the new-formed fibrous tissue is more diffusely distributed within the lobes and between the individual acini; here the islets are much more affected.

In a second paper Opie† describes an extremely important case of a negro woman, æt. 54, who died of diabetes without coma. The islands of Langerhans were the seat of a hyaline degeneration which had left the secreting portion of the gland unaltered; the islands were transformed into a hyaline homogeneous substance.

Such a case as this, coupled with the one already described, is conclusive proof of the dependence of at least some cases of diabetes upon disease of the islands of Langerhans, and brings the disease into line with others depending upon

* 'Practice of Medicine,' 4th ed., p. 422.

† 'Diabetes Mellitus,' p. 142.

‡ 'Centralbl. für Allg. Path. und Anat.,' 1900, vol. xi p. 202.

* E. L. Opie, "On the Relation of Chronic Interstitial Pancreatitis to the Islands of Langerhans and to Diabetes Mellitus," 'Journal of Experimental Medicine,' vol. v, January, 1901.

† E. L. Opie, "The Relation of Diabetes Mellitus to Lesions of the Pancreas," loc. cit., March, 1901.

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disease of ductless glands, such as myxœdema, exophthalmic goitre, Addison's disease, and acromegaly.

Since Opie's paper other cases showing affection of the islands have been published, and J. Dutton Steele* collects 35:

In 24 the pancreatitis affected the secreting portion of the gland, but much more intensely the islands.

In 6 hyaline degeneration of the islands was found.

In 4 chronic interstitial pancreatitis, with secondary and late involvement of the islands.

In 1 acute necrotic change involving the islands was found in a boy of ten years who died of acute diabetes.

The dependence, then, of some cases of diabetes on destruction of the function of the cell-islands of Langerhans, or to put it in another way, on the loss of their internal secretion, seems undoubted.

All observers seem to agree that in many cases of genuine diabetes no changes can be detected in the islands of Langerhans. Possibly further research may demonstrate the presence of change at present not recognised.

Excluding such cases, there are two groups of cases to be considered:

1. Those due to disease strictly restricted to the cell-islands, vide supra. The etiology of such is at present unknown.

2. Those dependent on disease of the cell-islands, the secondary result of a preceding pancreatitis.

The etiology of pancreatitis is fully entered into by Mayo Robson and Moynihan,† who state: "Chronic pancreatitis is quite distinct from the acute or subacute form, though it is probable that some of the latter cases end in chronic interstitial pancreatitis. As clinically seen, it probably usually arises by extension of the inflammation from a chronic catarrh of the pancreatic duct extending from a gastro-duodenal catarrh, though it is frequently associated with biliary or pancreatic lithiasis, or with gastric, pyloric, or duodenal ulcer." The explanation of the nature of the means by which this internal secretion of the pancreas affects the carbohydrate metabolism of the body is at present not attainable. Until the normal relations of this internal secretion to sugar metabolism are known, its pathological relationships must be still more obscure. In some way it is essential to health, so directing the sugar processes that the tissues receive and use sugar normally. Whether the process of absorption in the intestinal mucous membrane, or the glycogenic function of the liver, or the incorporation of the carbohydrate molecule into the proteid molecule, or the utilisation of the sugar by the tissues is interfered with, it is impossible to say. At some point failure occurs, and sugar is thrown on to the general circulation to automatically appear in the urine.

In severe cases, even in the absence of carbohydrate from the food, sugar is still excreted in the urine, and inasmuch as this is associated with an increased excretion of nitrogen in the urine and with rapid wasting, it would appear that the proteid tissues are being broken down and their carbohydrate group cleaved off. It is of course possible that there are several types of diabetes, but, in the probable event of such severe cases also depending on the pancreatic lesion, it would appear that the internal secretion must be responsible for not merely the ordinary transformation of sugar, but also for the integrity of the proteid molecule in the tissues.

Some conception of the delicate responsiveness of the pancreas as regards the nature and amount of its external secretion with varying foodstuffs is obtained by a study of the results obtained by Pawlow,* who finds that each sort of foodstuff determines the secretion of pancreatic juice richer or poorer in each particular ferment according as the food is rich or poor in proteid, fat, or carbohydrate. Thus a richly carbohydrate food evokes a pancreatic secretion rich in amylolytic ferment. If such exact relationship occurs as regards the external secretion, it may reasonably be expected that with a rich carbohydrate diet the internal secretion would be powerfully stimulated, as the absorption of much carbohydrate must necessarily involve great activity in the sugar metabolism after the sugar has passed through the intestinal mucous membrane. At the same time it must be confessed that the two

* "Chronic Interstitial Pancreatitis," 'Amer. Journ. of Med. Sciences,' July, 1902.

† 'Diseases of the Pancreas,' 1902.

* J. P. Pawlow, 'The Work of the Digestive Glands,' London (trans.), 1902.

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functions do not seem to necessarily go hand in hand, for pancreatitis of an extreme degree may occur when the external secretion must be greatly disturbed, and yet there may be no diabetes; and it is noteworthy that in such cases the islets of Langerhans have been found to be comparatively intact though surrounded by dense fibrous tissue.

The evocation of the pancreatic secretion is due to the entrance of acid chyme into the duodenum, and Bayliss and Starling * have recently shown that the contact of the acid with the epithelium of the duodenum and jejunum causes in them the production of a chemical body which they name secretin; this is absorbed by the blood and carried to the pancreas, where it stimulates the cells to secrete. This body is not a ferment. It might be expected that at the same time it would stimulate the cell-islands, and as it is easily obtained by scraping the mucous membrane of the duodenum and jejunum, it would not be difficult to determine whether after active experimental secretion has been produced by injection of large amounts of secretin the cell-islands show any change. Doubtless such experiments will be forthcoming.

Pavy considers that in the milder forms of diabetes, which he calls "alimentary," the fault is primarily in the intestinal tract, the cells of which fail to build up the sugar into the compound proteid molecule, with the result that too much is thrown into the liver—more than it can deal with, and that therefore sugar passes through into the general circulation and so into the urine. Such cases yield to dieting. In the severer forms, which he terms "composite," there is in addition a disintegration of proteid with the associated production of sugar and the acetone series of products, which we will consider shortly.

Such hypotheses would in no way be at variance with the abnormal absence of the pancreatic internal secretion—at any rate, so far as we can see,—for it is conceivable that whatever be the point at which the assimilation of carbohydrate fails, there may the function of the internal secretion be applied in health.

There are one or two other methods by which diabetes may be experimentally produced, which throw light on our knowledge of diabetes:

1. *Puncture diabetes.*—Bernard discovered that if a puncture be made in the floor of the fourth ventricle, glycosuria results. This glycosuria is due to a conversion of the liver glycogen into sugar, for at the end of the temporary glycosuria the liver is found to be glycogen free; and further, if the animal be starved for a period long enough to ensure its liver containing no glycogen, no sugar appears in the urine after the puncture. The condition is probably due to a stimulation of the vasomotor centre which is in close proximity to the spot, injury of which produces glycosuria; this stimulation disturbs the circulation in the hepatic artery, with the result of a glycæmia "due to the diminution of the amount of oxygenated blood passing to the liver through the hepatic artery, causing an excitation of the liver-cells and such consequent alteration in their metabolic activity as ordinarily accompanies excitation."*

This may be so, but of course the explanation would equally apply in the case of the pancreas, which might be stimulated in the same way and cause the glycæmia.

2. *Phloridzin diabetes.*—Phloridzin is a glucoside found in the root-bark of apple and cherry trees; if swallowed or injected under the skin a temporary glycosuria appears; after one or two injections it is found that the liver is absolutely free from glycogen. After the administration of such an amount as is known by previous experiments to render the liver glycogen free, a further dose will still produce glycosuria, and inasmuch as the sugar is associated with an increased excretion of nitrogen, it is clear that the sugar must have been derived from proteid. If phloridzin be injected into one renal artery the corresponding kidney at once begins to secrete sugar, from which it would appear that the drug acts primarily on the kidney. According to von Mering, the amount of sugar in the blood in phloridzin diabetes is not in excess, and may be less than normal.

Starling† states that—"We must therefore conclude that the primary action of the drug is on the kidney, exciting it to secrete the sugar normally present in the blood, and thus reducing the percentage of sugar below normal. The function of the liver is to keep the amount of sugar in the blood normal. As soon, therefore, as it receives

* 'Journal of Physiology,' vol. xxviii, 1902, No. 5.

* Schäfer, 'Text-book of Physiology,' vol. i, p. 927.

† 'Elements of Human Physiology,' 4th ed., p. 481.

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the blood which has been impoverished in sugar by the abnormal action of the kidneys, it will take of its store of glycogen, turning out sugar until the normal composition of this fluid is restored. And so these contending processes will go on, the liver pouring sugar into the blood and the kidneys at once excreting it out of the body. This struggle will continue until the hepatic store of glycogen is exhausted, and then, if the kidneys are still under the influence of phloridzin, the liver will attack the proteids in order to keep up the sugar supply, and there will be a rise of urea excretion in the urine which will run exactly parallel with the amount of sugar in this fluid."

Such a statement of course implies that the liver does send out sugar into the general circulation, and is opposed to Pavy's views. Pavy further denies that there is a diminution of sugar in the blood in phloridzin diabetes, and finds, as the results of analysis, that there is a distinct glycæmia.*

Whatever, then, may be the exact processes that go on in diabetes, the fact remains that the blood does contain an excess of sugar, and that in severe cases there is a disintegration of the body-proteid, with, as a result, the setting free of sugar in the blood. It becomes necessary to trace as far as possible the relationship existing between the presence of sugar and other substances in the blood and the various symptoms that may occur in diabetes. But first we must consider briefly the urine itself.

Urine.—The quantity is usually much increased, a common quantity being from five to ten pints. The specific gravity is high, and varies from 1020 to 1040 and over, though it must be remembered that a low specific gravity is not incompatible with the presence of small amounts of sugar. In appearance it is almost always clear and very pale, frequently with a greenish tint that is very suggestive of the presence of sugar. The reaction is commonly acid, and this despite the fact that the acidity may exist in the presence of a greatly increased percentage of ammonia.

We need not describe the ordinary tests for sugar, the most reliable being Fehling's and Trommer's, but especially the fermentation test. The tests depending on the reduction of cuprous oxide may give rise to error unless the presence of an actual precipitate, or at least a very definite reddish-yellow colour is insisted upon. Unless the copper hydrate produced on adding caustic potash be completely dissolved, a dark oxyhydrate is produced on boiling, which may be mistaken for the real sugar reaction. Further, when only little sugar is present such black precipitate may quite obscure the genuine reduction of the copper oxide by the sugar; it is therefore very important not to have an excess of copper sulphate.

It is also to be noted that after certain drugs, such as chloral and camphor, a body is found in the urine known as glycuronic acid, which may give the copper test, and after the administration of morphia, or after chloroform anæsthesia, sufficient dextrose itself may be present in the urine to produce a positive result with Trommer's or Fehling's tests.

Albumen is occasionally present, and if so may delay the production of the red precipitate.

The amount of urea is usually increased.

Before leaving the pathology of diabetes we will say a few words about diabetic coma, one of the most remarkable complications of diabetes, the nature of which has of late years become more fully understood.

Diabetic coma.—This condition is particularly apt to occur in patients whose urine contains a series of bodies, the presence of which in diabetic urine has long been known. Such bodies are acetone, diacetic acid, and oxybutyric acid, each of which has in turn been regarded as the cause of diabetic coma. As regards their origin, they are commonly supposed to be derived from breaking down proteids, but of late there has been a disposition to regard them as derivatives of the fats of the body. Acetone and diacetic acid are derivative products of oxy-butyric acid, and it is now generally held that coma is due to the presence of this latter body in large quantities in the blood and tissues, and Naunyn has given the term Acidosis to the condition produced.*

Another view is that in the process of proteid disintegration that goes on, the phosphorus and sulphur are oxidised into phosphoric and sulphuric acids. There is therefore a formation of acids,

* 'Journal of Physiology,' vol. xx, No. 6.

* See 'Path. Soc. Trans.,' 1900, "On the Pathology of Diabetic Coma," by Karl Grube.

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and these acids, by neutralising the alkalies of the blood, destroy its power of carrying away the carbonic acid formed in the tissues. For, in brief, the method of such removal is as follows:—The sodium carbonate of the blood in the presence of the carbonic acid of the tissues is converted into bicarbonate; this latter is conveyed to the lungs, where it gives up its carbonic acid and is reconverted into carbonate.

The phosphoric and sulphuric acids, by combining with the sodium, diminish the alkalinity of the blood and deprive it of its power of removing carbonic acid from the tissues. This therefore accumulates, and the patient is poisoned by it with the production of the condition of coma, a condition unaccompanied by cyanosis for the reason that the oxygen function of the red corpuscles is unaffected.

Confirmatory evidence of this possibility being correct is found in the results obtained by the experimental poisoning of animals by mineral acids; such was found as long ago as 1877 by Walter to lead to a condition closely similar to that found in patients dying of diabetic coma. It was also noticed that the amount of carbonic acid in the blood was enormously diminished, and this also has been found to be the case in diabetic coma. The presence in the urine of these three bodies, oxybutyric acid, diacetic acid, and of acetone, is, then, of serious import. Acetone can be tested for by Legal's test: a few drops of fresh solution of sodium nitro-prusside are added to the urine, and then a little caustic soda, giving a purple-red colour if acetone be present.

Diacetic acid is recognised by the deep red colour with ferric chloride solution. Oxybutyric acid is not so readily recognised, and requires the use of the polarimeter for its detection. It may be passed in enormous quantities, up to 100 or more grammes daily.

Ovariotomy during Pregnancy. — Paul Bar (*Bull. de la Soc. d'Obst. de Paris*, Nos. 5 and 6) illustrates the occasional tolerance of the pregnant uterus by reporting a case of cyst of the ovary whose pedicle underwent torsion at the fourth month, accompanied by the usual symptoms. Gestation continued undisturbed after removal of the tumour.—*Amer. Journ. of Obstetrics*, March, 1903.

CLINICAL LECTURES
ON
DISEASES OF CHILDREN.

LECTURE X.

RHEUMATISM IN CHILDHOOD.

Delivered at the London Hospital, November 22nd, 1902.

By ROBERT HUTCHISON, M.D.Ed., M.R.C.P.,
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GENTLEMEN,—I wish to bring before you to-day the subject of rheumatism as it manifests itself in children, or rather I should say, in early life. Now there are few diseases commoner than rheumatism in one or other of its many forms in childhood, and one may say also that there are few more serious, or in relation to which your responsibility is greater. Because rheumatism, you will remember, in early life is often the parent of cardiac disease in later life, and many people suffer from disease of the heart for a large part of their adult existence because they had an attack of rheumatism which was overlooked by the medical attendant in their childhood. That is why I want to impress upon you your responsibility in relation to this disease. It is a condition which, if properly treated, may be cut short, and if cut short, many consequences which would otherwise blight the life and activity of the subject of it may be prevented.

By rheumatism in children I mean something quite specific and definite. You know that the term in relation to adults is one which is used in a very vague sense. One talks of "chronic rheumatism" of a joint, for instance, without having any very clear idea before one's mind of the exact pathological state of that joint. Now in the case of children one does not talk in that loose way. One means by rheumatism an acute specific disease, which is almost certainly due to a specific micro-organism. Indeed, I think the modern view that acute rheumatism is due to a micro-organism receives strong support from the study of the disease in childhood. All micro-organismal diseases, all acute specific fevers, as you know, tend to be commoner in early life, and the great frequency of rheumatism in childhood is an *a priori* argument in favour of the view that it also is due to a specific

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micro-organism. Most people have now come round to that opinion, and they regard rheumatism as a morbid entity, as definite and precise in its pathology as typhoid fever or diphtheria. Now, though rheumatism may certainly be regarded nowadays as being due to a specific organism, you require for its production not merely the organism, but a suitably prepared soil. Clinical observation shows that all children are not equally subject to attacks of rheumatism. There are few diseases, indeed, in which heredity plays a more definite part. You constantly find when you inquire into the family history of children who are suffering from one or other manifestation of acute rheumatism that other members of their families have been similarly affected; and I think you can recognise when you meet them children who are so predisposed by heredity. I have often pointed out to some of you in the wards and in the out-patient room the typical rheumatic child, from whose facies you can say that it is prone to become the subject of this disease. These children are usually past the second dentition, for young children rarely suffer from rheumatism. They are dark rather than fair; their hair is dark, the eyes are dark, and they have long dark eyelashes. At the same time they have a peculiarly white skin and a very good complexion; they have a clear bluish-white sclerotic, and they have often very well formed, massive teeth, and particularly large, square, central upper incisors. They also exhibit very constantly what is termed a neurotic temperament,—that is to say, more than other children they are subject to minor nervous disorders. I think you will find it an advantage to have such a picture of the typical rheumatic child before you. It has occurred to me several times by recognising these characters to suspect rheumatism, or to be specially on the outlook for it, or to be able to say of a particular child that it will easily become the subject of rheumatism; and so to advise the taking of special precautions against infection.

Now unfortunately we do not know how those children become infected. We do not know definitely the path by which the rheumatic organism gains access to the body. We are unable to trace it as we can trace, for instance, the path of the tubercle bacillus. But I think there is at least a reasonable amount of evidence to show that infection takes place in perhaps the majority of cases through the throat. Now unhealthy throats are so common in children that one need never be surprised that the local resistance there is lowered; and I believe that a great many diseases could be prevented, and among them rheumatism, by a greater attention to the state of children's throats. I shall have occasion, however, when I come to talk of adenoids in infancy and in childhood, to speak a little more fully upon this subject. The organism, having gained access by the throat or by some other route of which we are ignorant, tends to manifest itself in certain tissues in children more than in others, and in particular the fibrous tissues and membranes are subject to its attack; I mean such membranes as the valves of the heart, the tendons, the sheaths of muscles, and the fibrous aponeuroses. These are the tissues which are most vulnerable, it would appear, to the attacks of the rheumatic bacillus. Now in this respect rheumatism as it shows itself in children differs considerably from the same disease as you see it in grown-up people, and it is much to be regretted that the ordinary text-book description of rheumatism is drawn from a study of the disease as it occurs in adults. Rheumatism ought to be described as it occurs in children, because it is in children that you see it in by far its most virulent manifestation. Now no one who has seen much of the disease in children will fail to recognise that the joint manifestations are often very trivial or altogether absent, so that had it been studied primarily in children it would never have been called acute articular rheumatism, which is the name usually given to it in text-books. That title is correct as regards grown-up people; in them the joint manifestations dominate the whole picture; but in children it is not so. In them rheumatism may invade the body, and almost pass over the joints altogether; or at all events the joint affection may be so trivial and transitory that it may be completely overlooked. On the other hand, it is an unfortunate fact that it is the valves of the heart more, perhaps, than any other part which tend to be the chief point of attack of the rheumatic poison in children, and for that reason the disease assumes in them a degree of danger and importance which is wanting in the case of the adult. Not only is the joint affection often exceedingly trivial in children, but you will often find that the degree of fever is extremely slight. Children suffering from acute

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rheumatism may have a rise of temperature amounting to only one or two degrees, and they hardly ever exhibit that dangerous rise of temperature which is spoken of as rheumatic hyperpyrexia. For some reason the whole febrile side of the disease is much less marked in the young. Indeed, some people go the length of saying that it would be more true to describe the endocarditis as the essential part of acute rheumatism in children, and the joint manifestations as mere complications, and that it is quite wrong to assume the attitude which one is apt to do and speak of the joint lesion as the essential thing and the endocarditis as the complication. The next point I want to emphasise is that the manifestations of rheumatism in early life are far more protean than in grown-up people. There are all sorts of forms of rheumatism in children which you may be apt to think are not rheumatic at all unless the fact is first pointed out to you. One may classify the chief manifestations of the disease as follows :—First, articular imflammation, the ordinary synovitis of joints; secondly, muscular or fascial rheumatism; thirdly, rheumatic nodules; fourthly, endocarditis, myocarditis, and pericarditis; fifthly, erythematous eruptions of various sorts; sixthly, chorea; seventh, pleurisy; and lastly, tonsillitis. I have not mentioned these rheumatic manifestations in the order of their importance. If one had done this one would have placed the heart manifestations first; I have merely put them on the board in series to show you how varied are the forms assumed by rheumatism in early life. Now these different manifestations of the disease may co-exist, or they may succeed each other in any order. Given a child predisposed to rheumatism, it may show at one time a few articular pains. These pains may pass off, and a few months later the child may have a little muscular and fascial rheumatism somewhere; later on perhaps it gets an attack of chorea, and that may be complicated by an eruption of rheumatic nodules and by some inflammation of the pericardium; or the child may have repeated attacks of tonsillitis, and occasionally fugitive erythematous eruptions, or even perhaps an attack of pleurisy; and in that way all these events may be spread out over childhood, one occurring at one time and one at another, but all being simply different manifestations of the one poison—namely, that of acute rheumatism.

I now want to look briefly with you at each of these members of the rheumatic series.

The *joint pains* I have already referred to. I have pointed out that they are often so slight as to be overlooked. But if you watch your cases of rheumatism carefully you will usually find *some* involvement of the joint at least at one or other period of the illness. It is very rare for a case to run anything like a prolonged course without there being at any rate some articular inflammation; but its degree may be extremely slight. Further, the inflammation of the joints is not so intense in its local manifestations as in the grown-up person; there is much less swelling and heat, hardly ever any redness, and there is comparatively little pain.

The *muscular and fascial rheumatism* of childhood deserves your special attention, not because it is in itself of importance, but because it should act to you as a danger signal. The pains of muscular and fascial rheumatism are commonly called, in the nursery, "growing pains." That, gentlemen, is a phrase which has crippled a great many hearts. Mothers and nurses are apt to regard growing pains as something physiological, something to be expected in growth. I need hardly say to you that that is an absolutely mistaken view. Those so-called growing pains are, in the great majority of cases, nothing more than the local manifestations of rheumatism in the muscles and fascia, and if they are neglected and the child is allowed to go about with them, it may very easily slide into endocarditis. A great many cases of heart disease come before you in later life in which you can obtain no definite history of rheumatism in childhood; and many of these, no doubt, are cases in which there have been these fugitive muscular pains, which have been so trivial that no notice has been taken of them until the heart has become seriously damaged. Those pains are particularly apt to affect certain parts of the body. I think you will find that the tendons of the hamstring muscles are more commonly involved than any other part, and that may lead you into errors of diagnosis. For instance, you may think that you have to do with a case of spastic paraplegia because the child walks on its toes, with the knees slightly bent, in order to spare the tendons; or you may expect some bone trouble or some affection of the knee-joint.

The appearance of *nodules*, the third mani-

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festation I have mentioned, is almost peculiar to early life. I think I have only twice seen rheumatic nodules in grown-up people, whereas in children they are of extreme frequency. Probably many of you are familiar with the appearance of these nodules; they are little things, varying in size from a pin's head to the size of a pea or a bean. They tend to occur under the skin where the bones come near the surface. Thus you find them over the olecranon and the patella, or on the spines of the vertebræ, down the centre of the back. You may discover them also forming a festoon around the edge of the scapula, or dotted along the occipital suture at the back of the skull. You may also sometimes find them over the tendons. Presently I shall show you by means of lantern slides some nodules in one or two of these situations. But I want to impress upon you that they require to be carefully looked for. They are often more easily felt than seen, and they are apt to escape your observation. They are characterised by being perfectly painless, which may surprise you, and by being extremely mobile. When you cut into them you find they consist of little nodules of fibrous tissues, the fibres being arranged concentrically round a core of fibrin. Those nodules are not in themselves of any importance whatever; it does not matter if there are little fibrous lumps under the skin here and there; but, like those rheumatic pains which we have been considering, they are of the greatest importance as danger signals, because they tend to go along with the heart complications, with endocarditis and pericarditis, and if you find rheumatic nodules coming out you may be fairly certain that there is progressive heart mischief going on. Dr. Cheadle says that when these nodules become really large they are practically equivalent to a sentence of death, because their significance as regards the state of the heart is so serious. You may compare them, if you like, to the vegetations which form on the edge of the valve in the heart. Just as in endocarditis of the mitral valve vegetations form along its edge, so in the case of nodules there are analogous fibrous nodules under the skin; but in the one case you speak of a vegetation and in the other case you speak of a nodule. Pathologically they are very much the same thing in the two cases. In the case of the nodules you may say they are vegetations which you can see and feel, while you may call the vegetations on the valves of the heart nodules if you like.

The fourth group of manifestations are those which occur in the *heart*. These, I need hardly say, are the most important of all. It is because their results are incapable of being entirely compensated for, because a valve once attacked is damaged irretrievably, that these forms of rheumatic manifestation are so serious. The commonest of them is endocarditis associated with pericarditis, and you may take it that in the majority of children who suffer from rheumatic heart affection these go together. You may not always be able to recognise pericarditis, but there is usually some degree of it present if there is endocarditis; and probably the converse is also true. You know that of recent years great attention has been paid in the pathology of the heart to the myocardium, and to the study of the changes which it undergoes in disease. Now a study of the myocardium in acute rheumatism shows that it also is apt to be the seat of damage. You find small-cell infiltration between the muscle-fibres; you find degeneration of the latter, and you find later a development of fibrous patches.

It is because of these three things tending to occur more or less together—endocarditis, pericarditis, and myocarditis—that some people prefer to speak of the heart affection of rheumatism as simply "*carditis*," meaning an inflammation of the heart in all its parts; and that is what you have to deal with in the majority of cases. But all the valves of the heart are not equally subject to attack in rheumatism. You may take it that in most cases the lesion which results from rheumatic heart affection in early life is mitral stenosis; and in the majority of cases also that is associated with more or less adhesion of the pericardium. Certainly in fatal cases these are the two changes which you find almost constantly: vegetations along the edge of the mitral valve, and more or less adhesion and fluid in the pericardium. Many children will escape those rheumatic manifestations as far as they affect the heart; and it will depend to some extent upon you how many of your patients so escape, because the better you are at detecting rheumatism the more easily you recognise its trivial manifestations, such as growing pains, and the more seriously and energetically you treat them the less will be the tendency for the heart to be affected; and by so

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doing you will prevent a great deal of subsequent cardiac disease in later life.

In the majority of cases you will find that the first attack of rheumatism does not permanently damage the heart. It is only after the second or third attack that the heart becomes the seat of lasting disease. It is true that in the first attack you may hear a whiffing systolic murmur at the apex, and you may therefore conclude that you have to do with damage of the mitral valve, which will go on to mitral stenosis or incompetence; and it is perfectly true that in such a case you *may* have to do with permanent damage of the valve. Yet there is no doubt that in many cases the murmur disappears entirely later on and the heart becomes, so far as you can tell by examination, perfectly healthy once more. But if there is a repetition of the attack for a second or a third time the damage to the heart is almost certain to become permanent.

I now pass on to consider *erythemata* as a sign of rheumatism. An erythema is, as you know, a red eruption, which appears like a blush over part of the skin, accompanied by more or less local heat, and which fades away again in a short time; and there are some particular forms of erythema which are almost peculiar to rheumatism. One of them is erythema nodosum; I think everyone will admit that it is always of rheumatic origin. It consists of an eruption of spots, slightly elevated, and usually more or less round or oval, which tend to appear particularly on the lower extremities, over the shins or calves, and are slightly tender. The skin over them is glazed, and they are often accompanied by a local inflammation of the ankle-joint. Sometimes there is a hæmorrhagic tendency in these cases, so that you get not merely erythematous patches but actual hæmorrhages into the skin, and to that form the term *peliosis rheumatica* is applied. Then you may get large extensive areas of erythema over the trunk, a condition which is often spoken of as *erythema marginatum.* You will find that it tends to occur in circular patches with crescentic outlines. It may involve almost the whole body at one time, and may be, and not uncommonly is, accompanied by rheumatic manifestations in the heart.

Of *chorea* I do not intend to speak in detail. It is a disease which you have plenty of opportunity of seeing in the wards, and it is hardly necessary for me to take up your time with a description of it. I shall only say that as regards its relation to rheumatism most people believe that in about 75 per cent. of the cases of chorea there is an undoubted rheumatic association. Some people say it is invariably rheumatic, because you must remember that chorea is only one event in the series. It may be the first event, and if you follow the course of that child out you may easily find that later on it develops so-called growing pains, or joint inflammation, or endocarditis. *Pleurisy* also I do not require particularly to describe, because we shall come to it in another lecture; but it is not a common manifestation of rheumatism, and when it is rheumatic in its origin it is probably usually of the nature of a dry pleurisy, or one without much effusion. Seeing that the pleura is in the nature of a large fibrous membrane, one would expect it to be more commonly attacked by the rheumatic virus than it is. But it is not nearly so susceptible as many other fibrous membranes. The reason for that I do not know.

Tonsillitis in its relation to rheumatism is a subject about which it is very difficult to speak definitely, because I do not think one can say that there is any given type of inflammation of the tonsil of which you can assert, simply by looking at it, that it is rheumatic in origin. On the other hand, no doubt, if you go into the history of children who have suffered from other manifestations of rheumatism you will often get a report of repeated sore throats. It may be that these are simply children with unhealthy throats; that there is follicular tonsillitis; and that the rheumatic poison gets access to a throat previously diseased. In purely rheumatic cases I think you will find that the inflammation is not confined to the tonsils, but tends to spread to the soft palate, and that it particularly tends to involve the tendons of the muscles, the tensor palati and the levator palati, and to produce pain in upward and downward movement. At all events one expects, here as elsewhere, to get the fibrous tissues involved rather than adenoid tissue, which is, as you know, the main constituent of the tonsil.

No matter which one of those manifestations has appeared, after it has lasted for even a comparatively short time you will find that the child gets into a cachectic state—what one may call the rheumatic cachexia. It is recognised largely by

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anæmia. The rheumatic poison tends more even than most toxins to destroy the red blood-corpuscles; and if you trouble to examine the blood of a child during an attack of endocarditis or pericarditis you will find a great loss of blood-corpuscles in a short time; in a few days their number may drop by a million. One has sometimes been able to recognise the development of complications in an attack of rheumatism by the sudden pallor which the anæmia produces. Constantly, when going round the wards, if you have a child who is suffering from a slight affection of rheumatism with fever, you will be able to tell if pericarditis has developed from the sudden increase in the pallor of the face. The fresh outbreak of the rheumatic poison seems to produce a fresh destruction of blood.

Lastly we will consider what the *treatment* of rheumatism should be. I need hardly say you should do all you can to prevent the disease developing in those children who are predisposed to it by heredity and by their diathesis. I say that because, although rheumatism is certainly due to a micro-organism, there can be no doubt that such agents as chill have something to do with its production, probably acting by diminishing the resistance of the tissues for the time being. See therefore that these children are properly protected from cold, that they are suitably clothed, and that in particular they are not allowed to get wet feet and sit in school in damp boots, which is, I believe, a very common cause of rheumatism in childhood. Be careful of the rheumatic child also in the matter of bathing. Over and over again one has known rheumatism develop after sea-bathing and after the injudicious use of cold baths. Cold baths are very excellent things, but you must not make a fetish of them; and many cases of rheumatism in early life are set up by the tendency to "hardening" the child by making it take a cold bath in the morning. The next point in the prevention of rheumatism is that you must take all the minor manifestations of the disease seriously. You have to insist upon the child going to bed at the first sign of the disease, no matter how slight and trivial it may seem to be. You may be inclined to think that a little aching in one joint or a little rheumatic stiffness about the muscles of the neck or the back is not worth paying serious attention to, but if you know the child to be predisposed to rheumatism you must insist upon it being kept in bed. If that were regularly done I am certain that a great deal of serious cardiac disease in later life would be prevented.

In regard to the treatment of rheumatism when it is actually developed, your chief care must be to protect the heart. The rheumatic pains are inconvenient and uncomfortable, and so is pyrexia, but they are not in themselves dangerous; whereas the least degree of endocarditis or pericarditis involves a serious risk. Further, thanks to salicylates, you have the joint affections under your control. It is unfortunate that salicylates seem hardly to touch the cardiac manifestations; in fact, some people think their introduction has increased the amount of cardiac disease rather than diminished it by enabling the subjects of rheumatism to get up and go about sooner than they would have been allowed to do in the old days. When the pains are over you are apt to think that the disease is over, and to overlook the fact that there is a slight endocarditis going on, which, if it becomes established, ends in permanent damage to the valves. So that what one has to preach about the treatment of rheumatism is the necessity of *rest*. One would sometimes like to put these cases in double long splints and keep them on their backs for six months! I am sorry to say that of necessity in such a hospital as this one has to set a bad example in this matter, for we cannot keep these cases in long enough; but you have to impress upon the parents that they absolutely must allow prolonged rest so as to prevent the heart becoming permanently damaged later on. If you insist upon prolonged and absolute rest, and at the same time use salicylates for the local affection, applying blisters over the heart when there are signs of pericarditis, you will do a great deal to prevent your patients becoming permanently crippled, and you will consequently diminish the amount of cardiac disease which one sees in grown-up persons.

Chinaphenin.—This combination of quinine and phenacetine has been tried by von Noorden during the last two years in a number of affections, including whooping-cough, typhoid fever, pneumonia, neuralgias, etc. It apparently possesses the virtues of each of its ingredients, while their drawbacks are neutralised. It was always well tolerated. —*Journ. A.M.A.*, March 7th, 1903.

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The Index and Title-page for Vol. XXI will be published with the next issue of THE CLINICAL JOURNAL.

A CLINICAL LECTURE

ON THE

ACTION OF ALCOHOL AND SOME OTHER POISONS UPON THE NERVOUS SYSTEM.

Delivered at the Middlesex Hospital, January 9th, 1903.

By H. CAMPBELL THOMSON, M.D., F.R.C.P.,

Assistant Physician to the Middlesex Hospital; Physician to Out-patients, Hospital for Epilepsy and Paralysis, Maida Vale.

GENTLEMEN,—On the last occasion upon which I had an opportunity of lecturing to you we considered the nature and causes of some of the more common paralyses of the upper extremity, and we then noted that not infrequently such paralyses were due to various poisons, such as, for instance, lead.

To-day I propose to speak of some of the more important poisons which may affect the nervous system, and to discuss the clinical symptoms which they produce and the changes in the nervous system that may be attributed to them.

One of the most common poisons which we meet with is alcohol when taken in excessive doses over a long period of time. The effects of chronic alcoholism upon the nervous system are very numerous, and hitherto have been studied chiefly from three clinical aspects, viz: (1) from the aspect of lesions of the peripheral nerves; (2) delirium tremens; and (3) chronic alcoholic insanity. It has thus been rather the custom in general hospital work to look upon cases of peripheral alcoholic neuritis as such only, and to pass lightly over the effects of alcohol on other parts of the nervous system. There may, of course, be such cases where there are no other symptoms, but it will be found that in most of those which find their way into the hospital wards there are signs that the peripheral nerves are not the only parts diseased, but that the brain has suffered as well. Mental deterioration often only temporarily shows itself, and confusion of ideas, loss of memory for

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recent events, sleeplessness, suspicion, and untruthfulness are among the more prominent symptoms that may be observed.

Most of you will have experienced the difficulty of obtaining an accurate clinical history from such patients; not only have they a very deficient memory, but they are unable to fix their attention and tend to wander from one subject to another. In the more acute outbursts of delirium tremens hallucinations and delusions are among the chief symptoms, and the patients are occasionally, unless carefully watched, driven to suicide by their mental terrors.

A recent contribution to this subject has been made by Sidney John Cole,* in which he records three cases of peripheral neuritis, all of which showed mental symptoms during life, and in all of whom after death extensive changes were found in different groups of neurons in the central nervous system, in addition to changes in the peripheral nerves.

Alcohol appears to produce these symptoms by a definite action upon the nerve-elements themselves. At first, no doubt, it produces functional changes which cannot be recognised by the microscope, but finally definite and unmistakable degenerations can be seen. In many stages the symptoms abate, and therefore presumably the lesions are, at any rate up to a certain point, recoverable; on the other hand, death may occur from the general nervous affection or from the involvement of some special nerves. The part which actual inflammation plays in the degenerative process is probably a small one, as recent investigations tend to show that degeneration is the predominating factor. In this respect it tends to resemble the degenerations produced as a late result of syphilis, to which I shall allude later. Another disease which is frequently associated with mental disturbances is typhoid fever. There are but few cases of typhoid fever in which there is not delirium at some period of the disease, but as a rule the delirium disappears as the fever subsides. Occasionally, however, delirium may be almost the first symptom, and this type has been called the pre-febrile delirium. Such cases are considered to be grave, for it is thought that the early delirium indicates a want of resistance of the nerve-tissues to the poison, or, on the other hand, it may be that the poison in those cases has some special affinity for the nerve-tissues. Then, again, cases occur in which the delirium persists after the temperature has fallen, and occasionally such mental changes may terminate in loss of memory or in actual insanity.

It is probable that in most cases where any grave mental deterioration follows typhoid fever, there is some predisposition on the part of the individual to nervous disorders; for fortunately such cases are not common, but they are nevertheless very important to bear in mind as a possibility. Dr. Mickle* has given a very graphic description and analysis of the different phases of thought and feeling which a patient who is suffering from typhoid fever may go through. In considering the cause of the delirium in typhoid fever we have to remember that the symptoms are due to a mixed infection, for there are products of other bacilli than typhoid absorbed from the alimentary canal, and although the typhoid toxin may take a share, it is probable that other poisons frequently predominate.

With a view to finding out whether the typhoid toxin produced any recognisable changes in the central nervous system, Mr. A. G. R. Foulerton and I examined several human cases and also carried out a series of experiments upon animals, an account of which was published in the 'Lancet.'† From these investigations we came to the conclusion that there was no evidence to show that typhoid toxin causes any definite changes in the ganglion-cells of the central nervous system of a nature that constant and definite structural alterations can be demonstrated by the Nissl method of staining. We, however, were careful to point out that it by no means follows from this that certain nervous symptoms may not be produced by the pure toxin, as, indeed, it is probable that they are, but if so they do not appear to be accompanied by any constant changes in the cells that can be demonstrated by the microscope.

Associated with diphtheria we have another important poison, which, like alcohol, shows a marked tendency to attack certain nerves. Thus in post-diphtheritic paralysis the soft palate is generally the first part to be affected, and the resulting symptoms are nasal voice and regurgitation of fluid through the nose owing to the inability of the

* 'Brain,' Autumn, 1902.

* "Mental Wanderings," 'Brain,' Spring, 1901.

† 'Lancet,' April 21st, 1900.

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paralysed palatal muscles to shut off the oral from the nasal cavity. After this the muscles of the eye are usually affected, with the result that there is difficulty in performing the act of accommodation, and squinting is also often a feature. But, like alcohol, diphtheria may also exercise a more general effect, and death may ensue from involvement of such important structures as the phrenic or vagus nerves, a point which it is important to remember when making a prognosis.

There is a considerable difference of opinion regarding the exact locality of the nerve-lesions in post-diphtheritic paralysis; some observers maintain that the changes are limited to the peripheral nerves, while others think the primary lesion is in the anterior cornua. F. E. Batten,* who has made important investigations in this subject, concludes that the dominant lesion is one of the peripheral nerves and not of the cell-body.

On the other hand, Rainey,† who has contributed a valuable paper on the question, found definite changes in the anterior cornua, and he concludes that diphtheritic paralysis is associated not only with changes in the peripheral nerves, but also with alterations in the spinal cord itself. He also considers that in the majority of cases the cell changes are antecedent to the changes in the nerves.

This difference of opinion is, however, not really a very great one, and it becomes less marked when we consider some observations by Mouravieff, which appear to show that the holders of both views are equally correct, and that the poison affects the whole of the lower motor neurons, but does not necessarily affect the cell-body and its branches simultaneously.

Mouravieff considers that the earliest changes are to be found in the cells of the anterior cornua of the cord; at first there is no alteration in the peripheral nerve-fibres, but later on distinct changes in the nerve-fibres may be found. By the time this peripheral nerve change is evident, the cells of the anterior cornua will have resumed their normal appearance, all the previously altered cells having either regained a healthy condition or having undergone atrophy and disappeared without leaving any trace behind.

Thus you see that it may happen, according to the times of observation, that one observer may find changes in the cells of the anterior cornua only; another may find at a later period changes in the peripheral fibres only; while a third may be fortunate enough to find changes in both cells and fibres. My own work on this subject, undertaken in conjunction with Mr. Foulerton,* is limited, and while our series of cases which we published led us to conclude that certain changes in the cells could be produced by diphtheria intoxication, our experiments were not sufficiently numerous to throw much light on the exact period of intoxication at which changes are most likely to be found.

Another important poison, the chronic effects of which you often see, is lead. I dealt with the results of chronic lead-poisoning, as far as the peripheral nerves are concerned, at some length in my last lecture,† and I shall therefore pass them over now, merely reminding you that the extensor muscles of the wrist and fingers are the parts most commonly affected, while more rarely the small muscles of the hand are the chief sufferers.

The action of lead is, however, by no means confined to the peripheral nerves, and those of you who have attended my out-patient department have recently had an opportunity of seeing a case in which the symptoms were all apparently cerebral in origin. The patient, a man æt. 70, has been a painter for fifty years, and has suffered with continual headaches for thirty years. He is generally constipated, has had one attack of colic two years ago, but has never had any paralysis. In addition to the headache, he has had for the past fifteen years occasional attacks of giddiness, often accompanied by loss of consciousness, which are no doubt attacks of minor epilepsy. His pupils are unequal and do not react to light. This case is, then, an example of the chronic action of lead upon the cerebral neurons.

We now come to another poison, that of the tetanus bacillus, the effects of which are fortunately not so very commonly seen. The bacilli gain access to the body by means of a localised wound, which is often comparatively trivial in nature, and then manufacture a toxin which shows a special affinity for the nervous system; and after a varying incubation period of a few days the effects of the

* 'Brit. Med. Journ.,' 1898, II, and 'Trans. Path. Soc. London,' 1899.

† 'Journ. Path. and Bacteriology,' 1900.

* 'Edin. Med. Journ.,' January, 1902.

† 'Edin. Med. Journ.,' June, 1902.

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poison are shown by the onset of tetanic spasms, which frequently begin in connection with the muscles of the lower jaw.

The relation between tetanus toxin and the nervous system is a most important and interesting one, for it has been shown that nervous tissue possesses the remarkable property of being able to neutralise tetanus toxin, so that an emulsion made by thoroughly mixing many times the lethal dose of toxin with a small quantity of either normal cerebral or normal spinal substance can be injected into a mouse without producing any injurious effect. It has also been shown that the cerebral substance possesses a greater power to neutralise the toxin than the spinal substance; and, indeed, given equal weights, the cerebral substance will neutralise about three times as much as the spinal substance.

At first it was thought that this neutralisation depended upon the formation of some antitoxin substance, but it was eventually shown that this was not so, there being a definite combination between the nervous tissue and the poison. The exact nature of this combination is uncertain; it may be a chemical action or it may take place by a process of imbibition, in the same way that a particular tissue takes up a stain.

It would appear that the incubation period of the disease corresponds to the time which must elapse before sufficient quantities have been taken up by the nervous system to produce symptoms.

Several observers have found definite changes in the cells of the cerebral cortex after death, and Mr. Foulerton and I* were able to corroborate the presence of these changes, and also we further considered that these changes were specific in character, definitely varying with the intensity and duration of the dose of the poison.

There are many other poisons which one might speak of, and notably syphilis in its relation to tabes and general paralysis. There is also the question of intoxication arising from the intestine and its possible influence in the causation of some varieties of mental disease; but time will not now permit any consideration of these interesting problems.

I hope, however, that I may have succeeded in showing you that it is a mistake to look upon the nervous symptoms in these cases from a narrow standpoint. We must endeavour to investigate the effects of substances such as alcohol and diphtheria upon the whole of the nervous system, and not allow their special selective action upon local parts to obscure their more general effects; for it is only by taking the wider view that more definite and complete knowledge will ultimately be gained.

* "An Investigation into the Nature of the Changes produced in the Nerve-cells of the Cerebral Cortex by the Action of Tetanus Toxin," 'Med.-Chir. Trans.,' 1900, vol. lxxxiii.

TWO LECTURES
ON
DIABETES MELLITUS.

Delivered at the Post-graduate College, West London Hospital,

By A. E. RUSSELL, M.D., B.S.Lond., M.R.C.P.,

Assistant Physician to the West London Hospital; Demonstrator of Physiology, St. Thomas's Hospital.

LECTURE II.

INTO the general etiology and symptomatology we cannot enter in any detail.

ETIOLOGY.—The disease, in the main, is one of adult life. Difficult as it is to assign any one definite cause, there are none the less several factors which do appear to have a definite relationship to the disease. Firstly, it has frequently been observed that hereditary influence may play an important part. All observers are agreed that the Hebrew race is more prone to it than other peoples. Prolonged mental exertion and anxiety appear to predispose to it. Excessive beer drinking may lead to its appearance. Injuries to or diseases of the head occasionally cause it, and here the causal relationship is established by the experimental results obtained by injury to the medulla, though, strictly speaking, such cases should be classed under symptomatic glycosuria rather than under diabetes. Injuries to the abdomen are known to have produced it, doubtless by means of disturbance of the pancreas.

Obviously the correct explanation of the different causes would involve an understanding of the methods by which the sugar function is disturbed. In the case of disease of the medulla this is clear. In at any rate the majority of other cases the correct explanation would lie in an understanding of the different ways in which the cell-islets of Langerhans could be affected, so that the disease might find its etiological explanation in those conditions which could lead to a chronic pancreatitis such as could be capable of setting up such affection, whether secondarily or otherwise. A primary affection of the cell-islets, such as has been demonstrated above, would obviously be of different origin to a secondary process the result of infection via the common duct. The association of diabetes

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with arterio-sclerosis suggests that the functions of the cell-islets might be interfered with by disease of the vessels within the pancreas, and it must be remembered that they are richly supplied with vessels and would therefore be peculiarly liable to have their function impaired by a deficient blood-supply. The condition of sclerosis of the kidneys obtaining in general arterial disease is strictly comparable with this. Enough has been said to make it clear that in whatever way the cell-islets may become affected the result may be the same, and the type of this disease, whether mild, or severe, or acute, would depend on the nature of the process involved. Thus one would not expect a slow degeneration, the result of chronic and progressive arterial disease, to produce an acute attack of diabetes; while any cause, probably infective, that might lead to a rapid inflammation or degeneration of the cell-islets, whether associated with or apart from affection of the secreting portion of the gland, would tend to result in a more rapid form of the disease. It is known to occasionally follow the infectious fevers, and it would be interesting to know whether in such cases the disease follows a rapid course, indicating a specific effect of the particular toxin. The marked tendency of the disease in children to assume a rapidly fatal course is also well established, and it is known that in children the disease has not uncommonly followed after an attack of one of the specific fevers.

Onset.—This is usually gradual. As a rule, until the patient's attention is drawn to the increased amount of urine he will have experienced either no symptoms of ill-health or at most some indefinite debility or slight loss of weight. With the increasing excretion of urine an increased thirst becomes noticeable, and frequently an augmented appetite. The loss of weight may be a very marked condition, and this despite the increased consumption of food; in older patients the tendency to loss of weight is much less prominent. The muscular system is almost invariably weakened. The temperature in the absence of complications is usually low and often subnormal. The tongue is usually dry, bright red, and glazed. There is a marked tendency to stomatitis. The digestive functions are usually unimpaired, but constipation is common.

Diabetes is a disease of many complications, and among the more important are:

Nervous complications. Peripheral neuritis.—This is presumably due to the presence of the sugar in the blood affecting the peripheral nerves, though the amount of sugar in the urine, and therefore in the blood, appears to bear no relation to the neuritis. The cases may be divided into the hyperæsthetic type, characterised by sensory disturbances, such as trigeminal neuralgia or sciatica; the motor type, with weakness of limbs and loss of knee-jerks; and thirdly, the ataxic type, which from its simulation of locomotor ataxy has been described under the name diabetic pseudo-tabes, the combination found in some cases of ataxy, loss of knee-jerks, pains in limbs, and even a perforating ulcer of foot being prone to cause errors in diagnosis. It must be noted that the knee-jerks may be absent in diabetes without any other symptoms of peripheral neuritis.

Coma.—The causation of coma has already been referred to. It is a very common termination of diabetes, occurring in twenty-eight out of forty successive fatal cases observed by Williamson. It may occur at any age, and especially in the young. Rarely it occurs within a few weeks of the onset of the disease. Certain conditions appear to hasten its appearance; thus undue muscular exertion has preceded it; sudden alteration of diet in the direction of absolute exclusion of carbohydrates may undoubtedly precipitate its occurrence, and similarly constipation. Its appearance may be heralded by epigastric pain, nausea, or vomiting; or a feeling of lassitude may develop into a gradually increasing drowsiness. More rarely headache, restlessness, and even delirium are the precursors. One of the most striking features is the condition aptly termed "air-hunger," with deep sighing respirations. The breath of such patients nearly always has the characteristic odour attributed to acetone, and in itself is almost diagnostic, but unfortunately some people are quite insensitive to this odour. The urine in coma commonly contains large numbers of finely granular casts.

In the remaining numerous complications to which diabetics are liable it is not always easy to correlate any very definite relationship between the symptoms and the presence of the sugar in the urine.

Cutaneous system.—Balanitis, pruritus, etc., the result of the irritation of the urine. Boils and carbuncles; these must be attributed solely to

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skin infection, and presumably are merely the expression of a lowered resisting power against the invasion of micro-organisms. A similar predisposition to septic processes is well seen in the great liability of patients with chronic nephritis to septic skin infection. Xanthoma diabeticorum, which occurs very rarely, appears to be more definitely related to the diabetic state, inasmuch as it may diminish or disappear with a diminished sugar excretion and return with the reappearance of the symptoms. Bronzing of the skin occurs very rarely, and then is usually associated with cirrhosis of the liver. Gangrene may occur—commonly in the lower extremities. This complication is almost certainly attributable to the arterio-sclerosis which is so frequently present in diabetes. In an interesting paper C. S. Wallace* strongly upholds this view, finding well-marked atheroma in twenty-four out of twenty-six cases of diabetic gangrene admitted during a period of eleven years into St. Thomas's Hospital. Wallace further notes the tendency of patients suffering from septic processes to glycosuria, and makes the interesting observation that individuals suffering from septic processes are often on the border-line of glycosuria, as evidenced by the fact that a very much smaller dose of glucose added to their ordinary diet suffices to produce glycosuria than is the case in healthy people. Perforating ulcer of the foot may also occur.

Pulmonary complications are common, particularly acute pneumonia and tubercular broncho-pneumonia. Gangrene is also well known.

Eyes.—Cataract is the most common complication, but retinitis and retinal hæmorrhages are found, though more rarely.

Mental Symptoms.—A tendency to melancholia is noticeable.

DIAGNOSIS.—Two things have to be determined: firstly, whether sugar is present in the urine; and secondly, if present, whether it really represents the progressive disease diabetes.

We will take Fehling's solution as one of the commonest and most reliable reagents. This should be boiled before the addition of the urine to see that it remains perfectly clear. If, then, after the addition of the urine and re-boiling, no reduction occurs, it may be accepted that no sugar is present. The presence of albumen, however, may delay the reduction so that boiling should be more prolonged, or, if any doubt remains, the coagulated albumen should be filtered off and the test applied to the filtrate. A yellowish-green decolourisation does not represent sugar, and may be due to excess of uric acid or to glycuronic acid; this latter body, which appears in the urine after the administration of certain drugs, such as chloral, camphor, etc., is simply an oxidation product of glucose which is itself capable of reducing an alkaline solution of cuprous oxide. Should any doubt remain it can be dispelled by the fermentation test. To apply this a small piece of yeast is put into a test-tube full of the suspected urine, and the whole inverted over urine or mercury; if more than the merest bubble of gas accumulates at the top of the test-tube, sugar is present.

One simple condition that is prone to give rise to the presence of sugar in the urine is lactation. During the first week or so of suckling lactose may be present in the urine in sufficient amount as to give a very definite reduction of copper oxide, but if this be borne in mind the error of attributing it to diabetes will be avoided.

When together with the presence of sugar in the urine we find other symptoms pointing to diabetes little room for errors in diagnosis arises, but when in the routine examination of the urine sugar is found without any other symptoms it may give rise to considerable difficulty, and especially so in insurance cases. For it appears to begin with that individuals differ very considerably as to their capacity for assimilating sugar; thus in the healthy the administration of two or three hundred grammes of sugar will cause a transient glycosuria. Further we know that there are many cases of the so-called alimentary type of diabetes in which a moderate restriction of sugar causes the disappearance of the glycosuria; between these two types there are people in whom, while no glycosuria exists with ordinary diet, a small excess of sugar in the food leads to glycosuria. Very fat people are prone to slight glycosuria, and such cases have been found in time to develop into genuine diabetes. Strumpell particularly notes the tendency to this glycosuria in obese beer drinkers. Lastly, glycosuria may be present in a varied group of diseases, such as diseases of and injuries to the head,

* "Gangrene complicated by Glycosuria," 'Lancet,' Dec. 23rd, 1899.

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exophthalmic goitre, cirrhosis of the liver, gout, acromegaly, etc.; but in such the prognosis would depend solely on the symptoms of the disease in question, and would be unaffected by the presence in the urine of small quantities of sugar.

There are many cases of diabetes, particularly in its early stages, in which, unless the urine be examined for sugar, the disease may escape detection from the want of those salient symptoms, the presence of which would at once excite suspicion of the disease. In this connection I cannot do better than quote some weighty remarks made by Strumpell:*

"Diabetes often exists for some time unsuspected even by the physician. It may therefore be well to name over the symptoms which may be the first to attract the patient's attention, and which should therefore always suggest to a physician the possibility of the existence of diabetes. They are: 1, languor and debility; 2, furunculosis; 3, pruritus pudendi in women, balanitis in men; 4, cataract; 5, sciatica, especially if bilateral; 6, impotence. We should be in the habit of examining the urine for sugar in all cases when complaint is made of indefinite symptoms, which cannot be explained without further evidence, particularly if the patient is fat or nervous, and also if he is elderly and shows signs of arterio-sclerosis. If symptoms similar to those just enumerated lead to an examination of the urine, and the result of this is ambiguous, it is advisable to have the patient partake of a meal rich in starchy elements, and to examine the urine thereafter, preferably by means of the fermentation test. If then no sugar is found, diabetes does not exist."

Several blood tests have been described for the recognition of diabetic blood. Williamson's is as follows:† Twenty cubic millimètres of blood, taken by means of the capillary tube of a Gower's hæmoglobinometer, are diluted in a test-tube with twice that amount of water. To this is added one c.c. of a 1-in-6000 watery solution of methylene-blue, and finally 40 c.c. of liquor potassæ. A control experiment is made with non-diabetic blood. The fluid in each test-tube has a fairly deep blue colour. Both are placed in a beaker of water, which is raised to the boiling-point and allowed to boil for four minutes without shaking, by which time the fluid in the test-tube containing the diabetic blood changes its colour to a dirty pale yellow. The fluid in the tube containing the normal blood may change in tint, but never loses its blue colour. If the tubes be shaken whilst heating, the decolourised methylene-blue is oxidised by the air and a blue tint may return to the fluid. Williamson has never found this test to fail in a very large number of examinations. In cases in which the urine cannot be obtained for examination, this blood test might be of considerable aid to diagnosis. It depends entirely on the presence of the dextrose in the blood, as dextrose is able to decompose methylene-blue with the production of a colourless reduction product.

Course, Varieties, and Prognosis.—Firstly, it must be remarked that the disease runs a much more rapid course in children than in adults. It may exist in a mild or in a severe form; in the former, the alimentary type of Pavy, the disease is seen in its mildest form. In such cases, by careful restriction of the carbohydrate food, the power of assimilating sugar may be considerably increased, so that, as Pavy points out, such patients may after treatment be able to deal with a much larger amount of carbohydrate food than before. In the severe type, named composite by Pavy, even after complete withdrawal of carbohydrate food the urine still contains sugar. In such patients the prognosis is of course of the worst. The older the patient at the onset the better the prognosis; in stout, middle-aged people the disease takes its mildest course.

In some cases the disease runs a rapid course, and may fairly be called acute, death occurring within a few months or even weeks of the onset. On the whole it must be looked upon as a fatal disease, although it may take a very chronic course. Signs indicative of short duration of life are the presence of pulmonary complications, marked wasting, gangrene, and the presence in the urine of the acetone series of bodies. Coma is humanly speaking a hopeless condition.

Treatment.—Assuming that the patient leads a well-ordered life, the treatment practically resolves itself into one of diet.*

The difficulties that may arise in diminishing or cutting off the starchy elements of diet are better

* 'Text-book of Medicine,' 3rd American edition, p. 763.

† 'Diabetes Mellitus,' p. 191.

* For a good account see 'Hutchison's Food and Dietetics,' p. 467 *et seq.*

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realised "when one reflects that most people are in the habit of obtaining at least one half, and sometimes even three fourths, of their total daily supply of energy in a carbohydrate form." In order to supply the deficiency caused by limiting the ingestion of starchy foods, these must be replaced by increased proteid and fats, and inasmuch as fats yield for the same weight two and a quarter times as much energy as proteids, the indication is to make as much use of fats as is possible in any dietary. In cases in which it is found that the urine becomes sugar free on cutting off the carbohydrates, such amount of carbohydrate food may be added to the diet as is found by experiment that the patient can assimilate without sugar appearing in the urine. If sugar is present even on the most rigid diet, all starchy food should be withheld, with this proviso, that in some cases the health of the patient is far better when a limited amount of starchy food is added to the diet, even though it does cause an increased sugar excretion. It must also be remembered that in severe cases the sudden adoption of a rigid diet appears occasionally to precipitate the development of coma.

As regards the dishes that may be taken or must be avoided by diabetics, reference must be made to the text-books.

Of other methods of treatment that by drugs is of little avail, with the exception that opium and codeia have a very distinctly beneficial action; of the two, opium is probably the better. It certainly relieves the distressing thirst to some extent, allays restlessness, and diminishes to some extent the loss of sugar.

A rational treatment of diabetes would be the administration of the active principle of the pancreatic cell-islets, and preparations of the pancreas have been tried, but so far with little result; nevertheless it is in this direction that success in treatment must be looked for.

The treatment of the ordinary complications presents nothing especial. In coma intra-venous injections of a 3 per cent. solution of sodium bicarbonate should be tried, at least three or four pints of fluid being injected. In the case of patients whose urine gives the red coloration with ferric chloride, and particularly if their breath should smell of acetone, the bicarbonate should be given by the mouth to the extent of three or four drachms daily. In some cases this appears to have had a decidedly beneficial effect.

A CLINICAL LECTURE
ON
"FIBROIDS OF THE UTERUS."*

By MARY SCHARLIEB, M.D., M.S.,

Physician for Diseases of Women, Royal Free Hospital; Lecturer on Midwifery, London School of Medicine for Women.

DURING the last three months we have had in our ward several interesting cases of so-called fibroid tumours of the uterus. In two of these cases we have found the formidable complication of double pyosalpinx, while two have been instances of "sloughing fibroids."

It occurs to me that it is profitable for us to consider in some detail the indications for operation in cases of "fibroid of the uterus."

Luckily fibroids are not as dangerous as they are common, but they are much more dangerous than is commonly supposed. It has been recently stated that their mortality, apart from operation, is 3 in 2,160,112 or 0·000138 per cent. ('Lancet,' January 20th, 1900). On the other hand, Mr. Stanmore Bishop, in the 'Medical Press and Circular' (February 14th, 1900), mentions eighteen cases of death from unoperated fibroids.

It is not possible to state the death-rate for fibroids not submitted to operation, for the sufficient reason that a large proportion are never discovered by their owners and that many known by the owner to exist are never mentioned to doctors. Consequently such fibroids do not appear in case-books, nor do they swell the number of deaths in the 'Registrar-General's Returns.' In another large percentage of cases fibroids are discovered accidentally, and as they cause no pressing symptoms are not heeded by the patient or by her adviser. It is as a rule only fibroids causing symptoms which impress themselves on the minds of patient and doctor.

Even of these tumours we have no accurate record of death-rate, because many patients who have fibroids die of some disease really, but not apparently, connected with the tumour. For instance, women die of heart degeneration, of pleurisy, or of kidney disease. Any one of these diseases may arise independently of the fibroid, or may be really

* Delivered to the Students of the London (Royal Free Hospital) School of Medicine for Women, February 2nd, 1903.

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traceable to it; but they appear in the death certificate under their obvious headings, and the fibroid, whether innocent or guilty, is not mentioned.

Under these circumstances we cannot know the percentage of fibroids existing in the female population, nor can we know how many cause death directly or indirectly. We must hesitate to adopt the proposed death-rate of 3 in 2,160,112, partly because of the numbers which escape detection even when they do cause death, and partly because each of us in his or her own little experience of ten, fifteen, or twenty years, and out of a *clientèle* of less than 2,000,000 patients, can remember several cases of death directly and evidently due to fibroids, and a larger number which apparently would have caused death but for surgical intervention.

Statistics are very useful in exciting our interest in, and in directing our attention to, facts; but directly we want to lean on them, and to make exact scientific deductions from them, we recognise, if we are honest, how difficult—nay, how impossible—it is to do so.

For instance, in 100 cases of fibroid submitted to operation the indications may be 200 or 300, because two or more indications were present in many cases, and we cannot say whether the hæmorrhage, the retention of urine, or the steady deterioration of health was the determining cause of operation.

What we as practical people want to know is not the general death-rate, high or low, but what advice we ought to give to our patient in each case that comes before us; and while we know that many fibroids need no treatment, and that some are amenable to drugs, it is also certain that a group remains in which operation is necessary.

At the meeting of the British Medical Association at Cheltenham, in 1901, we heard the views of many experienced and trustworthy practitioners, and we found, I am glad to say, a general concensus of opinion as to the indications for operations in cases of fibroids.

The discussion was opened by Dr. William Duncan. His views and those of the other speakers are briefly summarised thus:

Indications for operation in cases of fibroids.

Dr. DUNCAN.

1. Hæmorrhage { Menorrhagia. Metrorrhagia.
2. Pressure symptoms.
3. Rapid growth of the tumour.
4. Co-existing pregnancy.

Dr. ALBAN DORAN.

1. Rapid growth.
2. Softening of the tumour.
3. Pressure symptoms.
4. Hæmorrhage.
5. Pregnancy.

Dr. CHRISTOPHER MARTIN.

1. Rapid growth.
2. Cystic degeneration.
3. Malignancy.
4. Pressure symptoms.
5. Septic infection and sloughing of the tumour.
6. Severe hæmorrhage.

Mr. BOWREMAN JESSETT.

1. Hæmorrhage.
2. Pain.
3. Pressure symptoms.
4. Rapid growth of the tumour.
5. Fibro-cystic tumours.
6. Tumours which fail to diminish after the menopause.

Professor BYERS

Accepted Dr. Duncan's indications for operation and added that in his opinion fibroids were not so simple as they were formerly held to be. They were liable to degenerations, and in his opinion each case should be judged on its own merits.

Mr. HARRISON CRIPPS.

1. Cystic degeneration.
2. Sloughing of tumour.
3. Pregnancy.
4. Obstruction of bowels.
5. Appearance of patient.
6. Hæmorrhage.
7. Pressure symptoms.

Dr. HAWKINS AMBLER

Gave the same indications as Dr. Duncan, plus certain morbid conditions of the heart, which is, he said, frequently diseased in cases of fibroids.

Professor ALBERT SMITH.

1. Hæmorrhage.
2. Pressure symptoms.
3. Rapid growth of tumour.
4. Appearance in servants, etc.

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Mrs. Boyd.

Same as Dr. Duncan, *plus* appearance and convenience.

The late Dr. Aust Lawrence

Agreed with Dr. Duncan, but thought pregnancy less dangerous than he did. He had attended eight women in seventeen confinements complicated with fibroids with good results to mothers and children.

Of the twelve practitioners taking part in the discussion eleven were agreed as to hæmorrhage, pressure symptoms, and rapid growth being indications. Eight looked on pregnancy as a frequent indication for operation. Of the remaining indications, such important conditions as a sloughing fibroid, obstruction of the bowels, malignant degeneration, and septic conditions of the tumour were each alluded to by one person only. This was, of course, simply from inadvertence, for no indications could be more positive. Cystic degeneration of the tumour was mentioned by three only, but may have been included under the heading of "softening of the tumour" given by two other members.

One member, Dr. Hawkins Ambler, drew attention to the injurious effect of fibroids on the musculature of the heart, and considered the co-existence of heart trouble in many cases an indication for operation.

Mr. Bowreman Jessett thought failure of the tumours to diminish after the menopause an indication for operation, though he would probably not regard this as absolute in the absence of all other indications.

Several members—Mr. Harrison Cripps, Professor Alfred Smith, Mrs. Boyd, and I—held that a further indication for operation was to be found in the woman's incapacity to earn her living, and in the painful position of unmarried women, especially those in dependent positions, such as servants, shopwomen, and governesses. We also felt that in cases of broken health and incapacity women of all stations were entitled to demand relief if they desired it after a full and fair statement had been made to them of the dangers and inconveniences inherent in operations on fibroids. The surgeon should be careful to make the patient fully understand not only the risk to life, but other consequences of operation—for instance, sterility and a premature menopause in cases of hysterectomy.

The literature of "fibroids" is now very abundant, and the various authors are almost unanimous in advising operation where one or more of the usually accepted indications exist.

Thus Dr. Charles Noble writes: "I am not yet ready to accept the proposition that every fibroid tumour should be removed as soon as it is discovered; but I am convinced that this plan of procedure would be much better than the one that has prevailed up to the present time. Granted that the rule is that the subjects of fibroid tumours become invalids for many years, even if they do not lose their lives, it follows that, if they can be safely relieved of their tumours, this is urgently necessary to save them from weary years of suffering. It is difficult to estimate the actual danger to life of fibroid tumours, but it is not inconsiderable. If these tumours can be removed with approximately the same mortality as ovarian cysts, we have as an argument in favour of such removal the many years of suffering which thus will be prevented. At the present time hysterectomy is done only for the larger fibroids and for those which are directly threatening the life of the patient; yet in the hands of our best operators, under these conditions, hysterectomy is done with a mortality approaching 5 per cent. Were the indications for the operation extended and these tumours removed when still small, I believe that this could be done with a mortality of not more than 1 or 2 per cent. This being the case, the benefits of operation should be stated to all women having fibroid tumours."

Bland-Sutton, in his book on 'Tumours,' gives the following indications for oöphorectomy:

1. A myoma is the cause of serious and repeated bleeding producing profound anæmia; the bleeding is uninfluenced by rest and by the administration of ergot. When these troubles are not due to a pedunculated myoma projecting into the uterine cavity, and the menopause cannot be reasonably expected for two or three years, oöphorectomy should be performed; failing this, hysterectomy, if the anatomical conditions are favourable.

2. A myoma of moderate size in a woman between 30 and 45 becomes impacted and causes retention of urine at each menstrual period. Such a case is very suitable for oöphorectomy.

Mr. Bland-Sutton continues: "The following conditions demand hysterectomy:

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"1. A myoma rapidly increasing in size and extending high above the pelvic brim, and pressing on the colon so as to cause intestinal obstruction.

"2. A myoma rapidly increasing after the menopause.

"3. A fibro-cystic myoma.

"4. A myoma that has given little trouble suddenly begins to enlarge rapidly, accompanied by rapid pulse, high temperature, and signs of septicæmia. These signs indicate septic infection of the tumour. A gangrenous myoma should be removed without delay; occasionally a gangrenous myoma is too large to be removed through the vagina, and requires abdominal hysterectomy.

"5. The large pedunculated myomata which simulate ovarian tumours may be easily dealt with by transfixion and ligature of their pedicles (abdominal myomectomy)."

Mr. E. Stanmore Bishop says: "Not mortality alone, but misery, the vegetative existence, the hopeless waiting for a deferred menopause while the conditions change steadily for the worse, handicapping any possible future operation, are sufficient indications."

In his book on 'Fibro-myomata' (Manchester, 1901), after discussing the usual medical remedies, drugs and baths, Mr. Bishop sums up as follows:

"Prolonged medical treatment of fibro-myomata with a view to radical cure is useless. It wastes time, permitting the occurrence of complications which greatly increase the risk of subsequent intervention, and deludes the patient into delusive hopes of disappearance which are inevitably doomed to disappointment. This, of course, does not imply that various drugs may not be temporarily used until operation is practicable."

As to electrical treatment, he says: "The use of the continuous current is admissible in certain cases where hæmorrhage is the principal symptom, and where no contra-indication exists.

"1. It is useless in fibro-cystic growths, in subperitoneal fibroids, and in polypi.

"2. It is absolutely dangerous where there is any co-existing disease, especially inflammatory disease of the Fallopian tubes and ovaries; and such concurrent disease is of frequent occurrence. Meredith's analysis of 147 cases operated on by Tait showed 45 cases complicated by pyosalpinx, 43 by chronic ovaritis, and 2 by ovarian abscess.

"3. The introduction of the positive pole into the uterine canal, if it can be made to affect the whole endometrium, has a distinct hæmostatic effect; but it is doubtful whether this is greater than would be produced by any other cauterising agent.

"4. The internal use of the negative pole does without doubt produce a decrease in size of the hard multinodular fibroma, but this decrease does not always persist.

"5. It is extremely rare for any myoma to entirely disappear under electrical treatment.

"6. Puncture of the fibroid is always risky, sometimes disastrous, and the addition of an electric current does not appear to greatly modify the effect [of such puncture].

"The use of electricity holds a median position between that of medicine and that of surgical measures. It has the advantage that many do not shrink from its use as they do from anything which may be called an operation; whilst, undoubtedly in certain cases, it does produce a definite and reliable effect."

Operative treatment.—Some cases of fibroids do not require any treatment at all. Fibromata may, indeed, almost be said either to require nothing or to require electrical or surgical interference.

It may, I think, be accepted as proved that fibro-myomata, although scientifically styled *benign* in contrast to malignant growths such as carcinoma, are by no means harmless, but expose the patient to great and increasing risk—risk of life partly due to their own inherent conditions, partly to the changes produced in them as time elapses; whilst in those cases in which life is not directly threatened they deprive the patient of those years of her existence in which her powers of enjoyment and usefulness should be greatest;—that in certain well-defined cases electrical treatment by mild currents is extremely useful, but that the only certain remedy is by surgical interference; that delay in resorting to this unnecessarily imports into the case its most dangerous complications, such as necrosis, pyosalpinx, hydronephrosis, adhesions, and cardiac troubles, while it immensely and unnecessarily increases the dangers attending operation.

Dr. Champneys ('Lancet,' January 20th, 1900), says:

"Whether it is desirable or not to remove them (fibroids) depends upon the merits of each case.

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For instance, bleeding is a symptom which may urgently call for relief by operation, though deaths from hæmorrhage are extremely rare. A perpetually recurring severe loss of blood may ruin a life, even if it does not cause death, and may expose a patient to a greatly increased risk from many intercurrent diseases. It must be remembered also that long-continued severe hæmorrhage is apt to destroy the power of making blood; it destroys the mint as well as the bank. Pain, again, though very difficult to estimate in another, may be a correct indication. In this matter objective signs of suffering, such as loss of sleep and affection of the pulse during the pain, are to be carefully observed, and are of more value than graphic descriptions on the part of the patient. Pain may ruin a life, though it probably never causes death. Pressure symptoms, apart from pain, are often an urgent indication for operation. Dangerous pressure symptoms almost invariably affect the urinary tract and cause retention of urine, a condition which should not be allowed to continue as directly threatening life. A comparatively small fibroid impacted in the pelvis and causing retention, if it cannot be pushed up into the abdomen, should be removed without delay. A fibroid may be fixed in the pelvis by impaction, by adhesions, or by growing under the peritoneum. It is only when fixed by simple impaction that it can be pushed up.

"Rapid growth is also an urgent indication for operation, especially when accompanied by bleeding.

"Mere size may or may not be an indication for operation . . . the question here is usually one of convenience. Under these circumstances the proper course is to explain the real situation to the patient and let her choose. If she has to earn her own living she will probably claim, and justly claim, to be relieved of a burden which may interfere with her living. If well off she may, or may not, wish to run the risk of operation.

"A cystic fibroid will grow, and it should be treated as an ovarian tumour,—that is, it should be removed. In practice it will often be mistaken for an ovarian tumour. Malignant degeneration, though often talked of, is seen very rarely indeed. Its chief sign would be rapid growth."

The late Dr. Alfred Meadows said: "There is, in truth, no folly greater than to attempt the impossible, and no worse treatment of conscience and character than the habitual practice of unreality. . . . I am fully convinced that to persuade women for months and years to swallow gallons of medicine, mostly of a depressing and debilitating kind, in the vain hope that we can thereby bring about the absorption and removal of a hard fibroid tumour, . . . is not only unscientific, but unreal and dishonest. . . . Of late years our knowledge of the action of many drugs has greatly increased, and the clearer our insight becomes the more completely does it dispose of the false reputation which has been dishonestly acquired by the remedies usually given in these cases." Then he asks: "How can potassium bromide, known for its action on the nervous system, have the smallest influence in promoting the disintegration or absorption of a solid tumour? . . . Mercury is believed by some—I confess I am not of the number—to have the power of promoting the absorption of inflammatory products. What relation is there between this reputed property and the alleged power of absorbing the organised constituents of a solid fibroid tumour? I have used perseveringly the chlorides, iodides, and bromides of mercury even to the maintenance of slight salivation kept up for some time, but I must frankly own that beyond the mischief to the patient's constitutional powers, which they are well capable of inflicting, I have not observed any result, and most certainly I have never seen the very smallest diminution in the size of the growth. In iodide of potassium and iodide of iron I have just as little belief for the purpose in question." Then he discusses the claims of chloride of calcium, which has, he says, a most undeserved reputation, and points out that the natural process of calcification which it is desired to emulate is a consequence and not a cause of a previous starvation of the tumour by diminution or loss of its blood-supply; and that, moreover, mere addition of lime to the patient's fluids by no means insures its deposition at any point at which we may desire it. "I believe," he says, "that drugs are utterly inert in promoting the removal of uterine fibroids."

Dr. William Duncan, in a clinical lecture reported in the 'Lancet,' April 21, 1900, said:—"At the Berlin Congress of Gynæcologists in 1899 Gottschalk insisted on the degeneration of the myocardium frequently associated with fibro-myomata,

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and Freund said that diseases of the heart and blood-vessels are very commonly associated with them, and that they are on that account far less innocent than is generally supposed. Kelly says that "hyaline degeneration of the heart-muscle and arterial sclerosis are sometimes seen in old cases." Hoffmeier speaks of the frequency of heart disease in cases of fibro-myomata; he collected eighteen cases in which sudden death occurred from heart failure—three of fatty disease and fifteen of brown atrophy. You must ever bear in mind, gentlemen, the grave risks which a woman who has uterine fibroids runs in the event of her becoming pregnant. Abortion is liable to occur, and the prognosis is grave. Lefour collected thirty-nine cases of abortion with fourteen deaths, a mortality of 36 per cent. Parturition in cases of fibroid tumour is usually a matter of difficulty, often of grave danger. Of 147 cases collected by Sasserott, the forceps were required in twenty, with a loss of eight mothers and thirteen children. In twenty-one cases the placenta required manual removal, and of these thirteen women died. Collectively the maternal mortality was 53 per cent. and the infantile mortality was 66 per cent. Tarnier, out of six cases delivered by forceps, lost four mothers and four children, and five women with fibroids died before delivery. Some cases are not capable of delivery by the natural route. Sanger collected forty-three cases where Cæsarean section became necessary, and in these the maternal mortality was 83·7 per cent.; and Pozzi collected twenty-eight cases with a maternal mortality of 86 per cent. Johnston estimates that during pregnancy or labour one third of the mothers and more than half of the children die, and he recommends celibacy when the tumour cannot be removed. Taking all these facts into consideration, and forming an unbiassed opinion, I ask, is anyone justified in saying that "deaths from fibroids, apart from operation, are excessively rare"?

It would be easy to continue this exhibition of the very general consensus of opinion among those best qualified to judge, but no useful object would be served. It would perhaps be better to attempt to gather up the results of our investigation so as to make them available for application.

Hæmorrhage was long considered the most absolute indication for operation, although many authorities urged that it was seldom fatal. Repeated, persistent, and profuse hæmorrhage may not only be directly fatal, but it causes a steady progressive deterioration of health which makes the patient defenceless before the attack of any serious disease or accident; not only so, but the hæmorrhage may be sufficient to incapacitate her from the duties and pleasures of life. She may be condemned by it to an absolutely invalid existence. In considering this indication we are not to be guided by the amount of blood lost, but by the effect of the hæmorrhage on the patient and by her age. It may be right to encourage a woman of forty-eight years of age to wait a little longer and see whether the menopause may not be soon established, but it would be wrong to give the same advice to a woman ten years younger. Women vary greatly in their power of recuperation, and in some cases are able to bear repeated and profuse losses wonderfully well. In such cases they should be carefully watched, so that, should the recuperative power begin to fail, they may be relieved before irreparable harm is done.

Pain is, in itself, seldom an indication for operation, but as a symptom of impaction of the growth in the pelvic cavity, or of infective and other disastrous changes, it is of great value. Some years ago a patient of mine, Mrs. B—, came up from the country to consult me on some other trouble. She entered my room nearly double with pain and showing the anxious, pale, and sweating countenance we associate with an agony of suffering. She had left home quite comfortable, but on leaving the train began to experience this severe pain. On examination I found that one fibroid of the many she possessed had slipped down behind the uterus and was incarcerated in the pelvis. The whole condition of things resembled that of a retroverted gravid uterus. I pushed the intruding fibroid out of the pelvis, and the patient went away relieved. She could not wear a pessary, or a ring might have obviated a repetition of the accident. As it was, she soon wrote to me that she was never comfortable except when lying down, and eventually she decided that life under the circumstances was ruined and that she had better part with her fibroids *en masse*. The operation was entirely satisfactory to her and to me.

Pressure symptoms are usually indications for operation, but not always. Thus ten years ago I was consulted by a lady for frequent attacks of

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retention of urine. Her general health was good. She had a fibroid uterus reaching nearly to the umbilicus, and one lobe of the tumour was impacted in the pelvis. She had passed the menopause and was in good health. I found it was possible to push the mass out of the pelvis and to keep it out by means of a large-sized ring pessary. The weight of the tumour was supported by a good abdominal belt, and no operation was contemplated. I still see the patient at intervals; she enjoys very good health.

On the other hand, a married woman æt. 41, but looking very old for her age, consulted me for alternate incontinence and retention of urine. She had a large fibroid pressing on the bladder. In her case the tumour could not be pushed up, and for six years she had suffered until she was nearly worn out by her misery. In this case hysterectomy gave perfect relief.

Another woman, æt. 64, was suffering from alternate retention and incontinence, and her uterus was crowded out of the pelvis, being completely procident. The abdomen was filled with a large, soft tumour. This proved to be a fibro-cyst growing from the fundus uteri by a short, slender pedicle. The pedicle was divided and the tumour removed. No abdominal operation could have been less dangerous, and none more successful.

Among pressure symptoms we must remember interference with the rectum. The presence of an incarcerated fibroid causes mechanical narrowing of the lumen of the rectum and also interferes with its nerves; hence frequently severe constipation. It also interferes with the veins and causes hæmorrhoids. Neither constipation nor piles are immediately dangerous, but they tend to deteriorate health and sometimes add weight to other indications for operation.

Obstruction of the bowels is an absolute, but an infrequent, indication for operation; none occurred in the series of one hundred consecutive cases that I have just published,* but I have found several cases detailed in recent literature. The usual cause of such obstruction is the entanglement of a coil of intestine by the long pedicle of a subperitoneal fibroid.

Rapid growth of fibroid tumours is an indication for operation, because it generally means that some degeneration has occurred in the growth, such as cystic, mucoid, myxomatous, or inflammatory changes. Rapidity of growth is an indication which seldom occurs alone. It is usually associated with pain, often with pressure symptoms, and sometimes with a septic condition.

Pregnancy is a grave complication of fibroids, and although it is not an absolute and invariable indication for operation it always demands most careful thought; among the dangers involved are:

1. Abortion, which under these circumstances has the astounding mortality of 36 per cent.

2. Hæmorrhage after delivery, the tumour interfering with the normal contraction of the uterine musculature and so hindering the closure of the uterine sinuses.

3. Sepsis after delivery. This may be due to retention of portions of placenta and membranes which have not been detached and extruded as usual because the regular concentric shrinking of the placental site has been hindered.

Also it may be due to injury to the tumour or its capsule, either by the bruising caused by the uterine contractions or by the manipulations or instrumentation necessary to procure delivery. Thus only can we explain the wonderful mortality mentioned by Dr. William Duncan as occurring in the practice of such careful and able men as Tarnier. In six cases of labour complicated with fibroids and terminated by forceps he lost four mothers and four children, a mortality of 66·6 per cent. Nor are matters better when the labour is terminated by Cæsarean section; Sanger reports a mortality of 83·7 per cent. in forty-three cases, and Pozzi a mortality of 86 per cent. in twenty-eight cases. Probably in many of these cases ineffectual attempts at delivery *per vias naturales* had been made before the major operation was undertaken, and no doubt in many cases the vitality of the tumours had been injured by futile attempts to push them out of the pelvis.

The dangers arising from the association of fibroids with pregnancy commence early in gestation, increase during parturition, and are not ended with the completion of that process. Altogether we are told one third of the mothers and half the children perish (Johnston). The prognosis is best when the fibroids are subperitoneal and do not invade the pelvis. It is worst in the cases of

* 'Journal of Obstetrics and Gynæcology of the British Empire,' October, 1902.

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cervical fibroids, which usually act as mechanical obstructions to delivery, and can in no case escape injurious pressure.

Of course some cases are not fatal and some pass successfully through the ordeal. For instance, in a case sent to me by Mrs. Garrett Anderson, a lady who was known to have a fibroid married and soon became pregnant. Remembering the stimulating influence of gestation on fibroids, and fearing that the combination would prove disastrous, I advised operation. The patient and her husband were very anxious to have a living child, and went to Dr. Cullingworth for his opinion. He thought it possible to await the course of events. The patient went to term and was safely delivered.

The ethical indication also needs careful consideration, and whereas in a young unmarried woman appearance would count for much, and would sometimes be a valid indication for operation, the same importance could not be attached to it in a married woman. The question of incapacity for the duties and pleasures of life is important, and the decision of operation or no operation must be left to the patient, after a careful explanation of all the consequences of her decision.

The death-rate of operations for the removal of fibroids is constantly falling, and is about 5 per cent. in careful hands, provided that they do not attempt the impossible—for instance, the removal of huge tumours firmly welded to all the abdominal viscera, or operating in desperation on a patient who is evidently already septic and too feeble to endure the shock of a serious operation.

More patients are lost from undue delay than from too early interference. There is no doubt that not only do local conditions become more unfavourable, but that the general health deteriorates and the condition of the heart becomes steadily worse, so that the patient is operated on when the chances of success are seriously compromised.

Malignancy associated with a fibroid is, of course, an absolute indication for its removal. But not only is it an infrequent degeneration, but it is not often diagnosed before operation. The chief symptoms of malignant change are rapid growth, pain, and increasing severe hæmorrhage. This group of symptoms would indicate the desirability of operation in any case, but they may be signs of other degenerations besides that of malignancy. Foul discharge per vaginam may indicate malignant degeneration of the endometrium whether a fibroid be present or not; but when a foul discharge is associated with a fibroid it primarily suggests not malignancy, but sloughing or some other septic condition. It is, of course, an urgent indication for some form of operative treatment, palliative or radical.

I am very conscious that I have by no means exhausted the large and important subject of indications for operation in cases of fibroids, but I hope I have said enough to make it evident that the advice to be given in such cases is always an anxious responsibility, and that we should exercise as much care in dissuading patients from operation as in urging them to consent. There must be no foregone conclusion in our minds, and no one should be able to say of us: "Oh, consult A. if you think operation desirable, but take your case to Z. if you have advised the patient to wait awhile."

With all our care we shall make mistakes, and shall sometimes have the sorrow of knowing that we have waited too long and have allowed the patient to pass into a condition from which we are powerless to rescue her.

WITH DR. JAMES GALLOWAY AT THE POLYCLINIC.

(*Concluded from p.* 384.)

Peculiar Inflammation of the Skin Glands of the Face.

The patient who is coming in next is a girl already presented to the Dermatological Society of London, and whose case is one of exceptional interest, though I am afraid that at first glance you will not think there is very much to be seen. She came up to see me again at the hospital to-day, and I prevailed upon her to come and show herself here, thinking that the opportunity of your viewing the case was too good to be missed. She was sent to me by Dr. Leslie, of Bexley Heath, and her story is that the eruption commenced about five years ago. It rapidly affected symmetrically the portions of the face which you see now involved. The

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features of the eruption which you will notice are first of all marked erythema, a certain amount of desquamation, which varies and has varied very much under observation. When she came to see me first of all there was very little desquamation; there was only erythema and the lesion which I shall mention. The important lesion in this case is the formation of deep-seated cysts or nodules, which are apparently localised in the tortuous sweat glands or in their ducts, or possibly in the sebaceous glands. This goes on until finally—unless there happens to be a little accidental pustulation, as there is at the present time—there occurs a pitting, and therefore an atrophying of the skin. The lesions can hardly be called true cysts. As you know, there is a true cystic formation in the sweat glands or ducts, which is known as hydrocystoma, quite different from this case. In this case there seems to be very small, almost solid papules, deep in the skin first of all, and gradually appearing above the surface, and which finally leave little atrophied points. Now I do not know if you recollect having seen certain patients, usually young people, whose faces become pitted over with pinpoint like depressions, as a result of some inflammatory process which has passed over. Before the Dermatological Society of London these cases have come up not infrequently, and the exact origin of them has not yet been determined, nor has the character of the inflammation which produces this cribriform character of the skin been ascertained. But in this patient the process of destruction seems to be in its course. I would like you to look especially at the lobules of the ear. There a little suppuration has disfigured the patches, but there are the little elevated papules which I have already mentioned. In the region of the malar bone you will see the pitting going on. Remember that the process has been going on slowly for five years, that there has been no exudation or attempt at anything like eczema. How to classify it or define it I do not know. My colleague, Dr. Colcott Fox, drew my attention at the meeting of the Society to a picture in his private collection of a similar condition. Dr. Fox thought it was a peculiar form of inflammation of the sebaceous glands. But you all see that there is no comedo formation, and that there is nothing like acne. She gives no history as to its origin. She is in fairly good health, but she suffers slightly from dyspepsia, which may account for some of the flushing of the face which is apparent, but it has very little connection with the lesions. There is no telangiectasis, but there is well-marked erythema. It cannot be brought into line with any congenital defect, such as adenoma sebaceum. She has not had any treatment, except a little acid and gentian after meals for the indigestion of which she has complained. Dr. Fox suggests to me that it is a disorder of the sebaceous glands, and that I should use sulphur applications, as his case had been cured by this means.

Diagnosis of Acute Seborrhœic Dermatitis.

The next case is one in respect to which I will ask you to form a judgment without any indications from me. He was sent to me by Dr. Wilkin, an old student of the hospital, with a request for my opinion. The commencement of it dates four weeks back. The trouble commenced on each side of this young man's nose, and it has now spread, as you see, over the trunk, and also affects the lower extremities. I may say it does not affect the feet, and it diminishes as the ankles are approached. A suggestion which was made to me at first was that it might be syphilis; but on taking the whole series of events into consideration —the duration of the disease, the peculiar way in which it commenced, that is to say, symmetrically on each side of the nose, the large amount of exudation, the small amount of infiltration, the considerable amount of œdema, the destruction of the epithelium being comparatively slight, its distribution, spreading downwards on both the front and the back of the chest, the complete absence of lesions of the mucous membranes, the almost complete absence of adenitis—I came to the conclusion that it was almost certainly a somewhat unusual, certainly an unusually acute, seborrhœic dermatitis. The possibilities which we had in mind were first that it might be a papular and squamous syphilide, one which certainly should have been considered; pityriasis rosea, from which the exudation, the œdema, and the distribution served to distinguish it; and psoriasis had to be taken into consideration. Finally we were left with the possibility of an acute circinate seborrhœa corporis, and I think that is the actual state of affairs. But it is a sufficiently unusual case to make it worth while bringing it before you.

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